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I declare that this report is my own original work and that the contributions of others have been duly acknowledged..............................

Dedication

This thesis is dedicated to all those who have struggled through postgraduate research and academic adversity, yet who have managed to persevere to achieve the final goal. Also this thesis is dedicated to my loving and wonderful parents, without whom I could not have succeeded in completing my postgraduate education.

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The effects of alcohol consumption and expectancy on anxiety in men and women.

Literature Review

Abstract

Experimental research investigating the effects of alcohol consumption on anxiety has been equivocal, and alcohol consumption has been found to increase and decrease anxiety and also to have no effect on anxiety. These results are further complicated by evidence of significant sex differences in the effects of alcohol expectancy on anxiety. In this review, several theoretical models of the effects of alcohol are evaluated for their utility in explaining the relationship between alcohol and anxiety. Then, research utilising the balanced placebo design to investigate the relationship between alcohol and anxiety is reviewed, and the influence of pharmacological mechanisms, alcohol expectancy, and sex differences discussed. Finally, issues involved in defining and measuring anxiety are considered, and methodological limitations are presented as partially explaining the perplexing results in the area.

The general aim of this review is to evaluate the effects of alcohol consumption and expectancy on anxiety in men and women. Several theoretical models of the effects of alcohol will be presented and examined for their ability to elucidate the relationship between alcohol and anxiety. Experimental research which has utilised the balanced placebo design to examine the relationship between alcohol and anxiety will be discussed, and the influence of alcohol expectancy and pharmacological mechanisms will be examined, as will sex differences in the effects of alcohol expectancy on anxiety. Finally, various methodological concerns relevant to interpretation of the literature will be presented.

It is widely assumed by the layman that a primary function of alcohol is to reduce tension and alleviate stress. This concept is widely known in the experimental literature as the tension reduction hypothesis (Conger, 1956). However, despite much empirical investigation, the relationship between alcohol consumption and anxiety is neither well characterised, nor well understood. Alcohol consumption has been demonstrated to reduce anxiety, to increase anxiety, and also to have a neutral effect (Young, Oei, & Knight, 1990). Much of this experimental confusion may be attributed to the differential influence of pharmacological mechanisms and alcohol expectancy factors.

Most early research assumed that alcohol predominantly affected tension or anxiety because of its pharmacological impact upon perceptual, cognitive, and/or motivational states (Hull, 1981). Alcohol, as a drug, is classified as a central nervous system (CNS) depressant which has non-specific actions on the CNS. More recently, it has been discovered that in addition to non-specific and disorganising effects, alcohol alters the function of an important inhibitory neurotransmitter known as gamma amino butyric Acid (GABAb). GABAb is a major inhibitory neurotransmitter in the brain and GABA containing neurones are widely distributed, being relevant to most CNS functions. Alcohol potentiates the action of a particular type of GABAb receptor, increasing its responsivity to the neurotransmitter, GABAb. Hence, increased GABAb transmission results in relaxation and reduced tension or anxiety (Kalat, 1995; Carlson, 1994). At this time, any alteration in tension or anxiety following alcohol consumption was attributed predominantly to pharmacological mechanisms.

However, since the 1970s attention has increasingly been given to the role of cognitive processes, known as expectancy factors in mediating the effects of alcohol (Bandura, 1985). The term expectancy refers to an intervening variable of a cognitive nature. Whether explicit or implied, this cognitive variable is understood to be knowledge (i.e., information, encodings, schemas, or scripts) about relationships between events or objects in the real world. The expectancy, rather than attitude or belief is invoked when the author refers to the anticipation of a systematic relationship between events or objects in an upcoming situation. The relationship is understood to be an if-then variety; if a certain event or object is registered then a certain event is expected to follow. Researchers usually intend a close linkage between the cognitive expectancy and the antecedent stimulus and consequent behaviours, although the relationship is often not clearly specified (Goldman, Brown, & Christiansen, 1987).

In laboratory work using the balanced placebo design (Marlatt, Demming, & Reid, 1973; Marlatt & Rohsenow, 1980), alcohol expectancy refers to the participant's belief that he or she has received an alcoholic or a non alcoholic beverage (Critchlow-Leigh, 1989). Research has shown that alcohol expectancy, or the belief that one has consumed alcohol, irrespective of actual drink content, may be sufficient to lead to disinhibition of behaviour or alteration of affective states (Hull & Bond, 1986). However, the significance of alcohol expectancy is more complex than simply the belief that one has been drinking.

Alcohol expectancy (i.e., the expectancy of ingesting alcohol) may be differentiated from the beliefs that people hold about the effects of alcohol, known as outcome expectancies. The nature of the relationship between alcohol expectancy and outcome expectancies is uncertain, although researchers generally accept that alcohol expectancy effects are directly linked to, and, are mediated by the content of outcome expectancies. For instance, Stacy, Newcomb, and Bentler (1991) posit a moderating effect of alcohol beliefs, suggesting that expectancy accessibility moderates the association between beliefs and alcohol related behaviour. Similarly, Goldman, Brown, Christiansen, and Smith (1991) assert that the content of the expectancy (i.e., the beliefs about the effects of alcohol on moods and emotions), and also the consequences of intoxication, have a mediating influence on the direction of expectancy effects. For example, independent of the knowledge regarding the contents of a drink, one may hold certain beliefs or expectations that drinking alcohol will have certain consequences (e.g., such as, increased friendliness and reduced tension). It is one thing to know or believe that one is or is not drinking alcohol; it is yet another to examine the meanings of drinking and the effects that individuals might expect.

THEORETICAL MODELS OF THE EFFECTS OF ALCOHOL

There are a number of theoretical models of the effects of alcohol which attempt to explain the empirical relationship between alcohol and anxiety, and which place differential emphasis on the importance of pharmacological mechanisms and cognitive expectancy factors. The tension reduction hypothesis (Conger, 1956) and the more contemporary stress response dampening model (Sher, 1987) will be discussed as specific, but restricted models which assert the predominance of pharmacological mechanisms in the mediation of anxiety following alcohol consumption. These models suggest that alcohol generally reduces anxiety and only in certain circumstances can alcohol increase anxiety. However, as more contemporary research suggests that there is no simple relationship between alcohol and anxiety, the social learning theory of alcohol use (Abrams & Niaura, 1987) will be offered as a broader and more comprehensive model for explaining the relationship between alcohol and anxiety.

Most research on the relationship between anxiety and alcohol has involved an investigation of the validity of the tension reduction theory of the effects of alcohol (Conger, 1956). This hypothesis contends that alcohol serves to reduce tension or anxiety, because of the pharmacologically depressive, or tranquillising effects it has on the central nervous system. Consequently, due to its tension reducing properties, alcohol consumption is reinforced. Several pharmacological mechanisms could be hypothesised, including a direct pharmacological effect on physiological responsiveness, with attenuated responsivity resulting in the inference of lower levels of anxiety. An alternative explanation may be that the effect is an indirect result of the effect of alcohol on a cognitive process such as increased distractibility (i.e., making the person attend less to the stressor), or altering evaluation of the level of the threat (i.e., making the person feel the stress is less severe (Levenson, Sher, Grossman, Newman, & Newlin, 1980).

Despite the array of research conducted with animals and humans(Keane & Lisman, 1980; Abrams, Lipscomb, & Wilson, 1980; Brick & Pohorecky, 1985), as tests of the tension reduction hypothesis, it remains empirically unsubstantiated. Rather than supporting the Tension Reduction Hypothesis, empirical research has illustrated the tremendous variability in the effects of alcohol on anxiety. Contemporary evaluations of the tension reduction hypothesis suggest a huge divergence between apparent faith in this model and the quality of evidence to support it. The main proposition of this model that alcohol consumption decreases anxiety and that in turn it is consumed for its tension reducing properties is not adequate to account for the variety of experimental effects seen. The model is global and broad and takes little account of important factors, such as situational and individual influences.

Cappell and Greelly (1987) suggest that the tension reduction hypothesis is not tenable as a single factor explanation of the effects of alcohol, but is valid only within a relatively circumscribed portion of the dose dependent curve, and makes its greatest contribution as a crucial component of more complex models such as the stress response dampening model (Sher, 1987).

The more contemporary stress response dampening model (Sher, 1987) bears close resemblance to the tension reduction hypothesis, and is based on empirical findings that the physiological stress response, and especially cardiovascular responding, is dampened by alcohol. Sher (1987) provides a psychopharmacological approach, in which the effects of alcohol are mediated by pharmacological mechanisms affecting central mechanisms in the brain sensitive to aversive stimuli.

Sher (1987), in keeping with the tension reduction hypothesis, argues that alcohol, at sufficient doses, dampens the physiological stress response, subjectively alleviating stress and thereby reinforcing drinking in similarly stressful situations. In contrast to this fundamental tenet, Sher (1987) does not assume that alcohol consumption universally results in stress dampening or anxiety reduction, or that all stressful situations will tend to elicit drinking behaviour. Real or imagined negative consequences of intoxication (i.e., impaired cognitive or behavioural performance in a social interaction) might increase anxiety. It is suggested that in some situations alcohol consumption is likely to be punished or proscribed and in contexts such as these the model does not assume that alcohol consumption will reduce stress. Therefore, it appears that the stress response dampening model allows for evidence that alcohol consumption may have anxiogenic or anxiolytic effects. Nevertheless, this allowance is restricted as the major tenet of this model is that given a sufficient dosage, alcohol will result in decreased stress responsivity.

Unfortunately, there remains some controversy regarding the nature of a "sufficient dose". Sher and Levenson (1982) suggest that a relatively large dose of alcohol (approximately 1g/kg) is necessary for demonstrating cardiovascular response dampening effects. However, more recent data suggests that significant stress response dampening effects can be seen at lower doses (e.g., 0.85g/kg) and that some individuals demonstrate significant stress response dampening effects as low as 0.425g/kg (Sher & Walitzer, 1986). In response, Sher (1987) suggests that the minimum dosage necessary to demonstrate a dampening of stress responsiveness is a function of a number of variables, including the time since drinking (i.e., the blood alcohol concentration and rate of change in BAC) and the nature and severity of the stressor.

Unfortunately also, the reliability of self reported stress response-dampening effects is poor. Although alcohol consumption often leads to decreased report of anxiety (Rimm, Briddell, Zimmerman, & Caddy, 1981; Polivy, Schueneman, & Carlson, 1976), a number of negative findings at several dosage levels demonstrates that the effect is not robust across a wide variety of experimental situations (Abrams & Wilson, 1979; Sher & Levenson, 1982). Finally, Sher (1987) makes no attempt to account for the influence of alcohol expectancy seen in the relationship between alcohol and anxiety.

A wide variety of theoretical perspectives are available that make a serious attempt to account for significant expectancy effects. Shapiro and Norris (1978) discuss some of these theoretical accounts including contributions of constructs such as suggestibility, demand characteristics, transference effects, cognitive dissonance, classical and operant conditioning and social learning theory (Abrams & Niaura, 1987). Since a discussion of all these accounts is beyond the depth of the present paper, emphasis will be placed on social learning theory.

Incorporating the importance of personal experiences and learning history, in the development of alcohol outcome expectancies (i.e., alcohol related beliefs) is integral to the social learning theory of alcohol use provided by Abrams and Niaura (1987). This interactionist theory is a broad but comprehensive theory, providing a set of social learning principles for alcohol use and its effects. social learning theory emphasises the importance of alcohol related beliefs and expectancy, whilst allowing for the accepted influence of pharmacological mechanisms. Social learning theory also provides adequate explanations of sex differences seen in the alcohol and anxiety literature. Furthermore, as will be discussed below, it supplies an adequate explanation for the development of alcohol related beliefs and expectancies, and consequently, how the content of these beliefs may mediate either an anxiolytic or anxiogenic effect.

Social learning theory broadly suggests that alcohol related beliefs (i.e., outcome expectancies) may interact with the pharmacological mechanisms to determine, in part, intoxication experiences. This is salient because it accounts for experimental research showing significant effects of both alcohol expectancy and consumption on anxiety. Abrams and Niaura (1987) further assert that available research indicates that expectancy set, especially when low doses are consumed, is a factor that cannot be ignored in theoretical explanations of drinking and behaviour when intoxicated.

According to social learning theory learning to drink is an integral part of psychosocial development and socialisation within a culture. Beliefs, attitudes, and expectancies concerning alcohol are formed vicariously through socialisation very early in life, prior to actual experiences with alcohol. This occurs primarily through the social influences of culture, family, and peers and may be exerted indirectly through their attitudes, expectancies, beliefs, and/or directly by modelling alcohol consumption and social reinforcement for drinking.

In support of this, Spiegler (1983) has shown, that, by the age of six, children have already clearly established perceptions of social drinking norms for men and women. Patterns of drinking behaviour, including drinking under circumscribed conditions or drinking in a large variety of circumstances, and in response to monotony or stress, are modelled by family and peers. From an early age individuals learn how alcohol is used, and in what situations and what behaviours are permitted when one is intoxicated (MacAndrews & Edgerton, 1969).

Direct experience with alcohol is believed to crystallise these vicariously learnt beliefs, attitudes, and expectancies (Christiansen, Goldman, & Brown, 1985). Bandura (1969) states that after initial exposure to alcohol use, the individual experiences directly the positive reinforcing effects of alcohol's stress reducing properties. For example, when tension reduction, mood enhancement, or better social interaction is experienced while drinking, this direct experience reinforces the belief that alcohol helps in social interaction situations, although these effects are mediated strongly by socially learned expectations, cultural norms, and role models. Thus, the short term effects of alcohol work as a self fulfilling prophecy.

As aforementioned, individual beliefs are believed to mediate expectancy effects of alcohol (Young et al., 1990) and differences in alcohol related beliefs may explain the inconsistency in expectancy effects on anxiety. For instance, if one holds negative beliefs about alcohol, due to aversive past experiences, alcohol expectancy in a laboratory situation is likely to have an anxiogenic effect. Conversely, if positive alcohol related beliefs are held, then alcohol consumption is more likely to have an anxiolytic effect. These effects are mediated strongly by socially learned expectations.

social learning theory is perhaps the most useful and comprehensive model available, for explaining the effects of alcohol consumption on anxiety. It is superior to others presented because it considers the bio-psycho-social nature of alcohol related changes in behaviour and affect. The model acknowledges the inevitable influence of pharmacological mechanisms, but emphasises greatly the importance of cognitive variables such as alcohol related beliefs and expectations, in the mediation of alcohol related changes in affective states and behaviour. It also provides an explanation for the development of individual beliefs and expectancies about the effects of alcohol use and how this may produce anxiogenic or anxiolytic effects following alcohol consumption. Finally, social learning theory accepts that, due to the multitude of influences that may affect the social drinker, simple conclusions about how alcohol affects anxiety are not tenable.

THE BALANCED PLACEBO DESIGN AND METHODOLOGICAL CONCERNS

In order to investigate the effects of alcohol expectancy and consumption on anxiety, researchers have developed and used the balanced placebo design (Marlatt et al., 1973; Marlatt & Rohsenow, 1980). This design allows for the independent variation of alcohol expectancy (i.e., by manipulating instructions concerning the beverage being consumed- told alcohol; told no alcohol) and the actual pharmacological properties of the drink itself (i.e., given alcohol, given no alcohol). In this 2 x 2 design, in which beverage content and expected beverage content are factorially crossed, half the participants are told they will be receiving alcohol, and the other half are told they would be receiving a non-alcoholic beverage. Within each group, half receive a non-alcoholic drink and half receive an alcoholic drink.

A between subjects design has been utilised and thus, the following four groups are involved: told alcohol/received alcohol, told alcohol/received no alcohol, told no alcohol/received no alcohol, told no alcohol/received alcohol. By factorially combining instructions pertaining to the beverage content and the actual beverage administered, investigators have the capability of determining whether an experimental effect is the result of the beverage administered, the participant's expectancies, or the interaction of the two factors (Bradlyn & Young, 1983). Hence, using Analysis of Variance, partitioning the main effects allows for disentanglement of the drug effect from the effects of its corresponding expectancies (Knight, Barbaree, & Boland, 1986).

In order to sustain the validity and utility of the balanced placebo design, a number of assumptions must be met. The most critical assumption is that the beverage deception effectively determines expectancies. This necessitates that the instructions given the participants lead to particular expectancies, and also that changes in affective state, behaviour, and/or physiology can be attributed to this intervening variable. Participants must believe their initial instructions regarding the content of the beverage, and also report similar experiences regardless of the beverage they were actually given. A second assumption is the requirement that blood alcohol concentration has little, if any effect on subsequent assessment of the beverage deception (Bradlyn & Young, 1983; Marlatt and Rohsenow, 1980).

In a related fashion, the validity of the balanced placebo design necessitates the participant's naivete with respect to the alcohol and expectancy manipulations . To maintain naivete, it is often necessary to disguise the presence or absence of alcohol in the beverage, utilising various techniques. Most studies also use a 5:1 ratio of tonic to alcohol as this level of alcohol has been determined to be undetectable by taste in this ratio (Bradlyn & Young, 1983; Knight et al., 1986).

Marlatt and Rohsenow (1980) report mixing the drinks in front of the participant, administering a breathalyser test and providing false feedback appropriate to the expectancy instructions given, rinsing with mouthwash or using peppermint water (to mask the taste), and smearing a few drops of alcohol on the rim of the glass (i.e., told alcohol/ given no alcohol condition). If, for example, the drinks are brought to the participant already prepared, a participant has no evidence other that the experimenter's word as to the nature of the drink being served. A sophisticated participant may doubt the sincerity of this information, becoming more attentive in the search for other cues, perhaps paying closer attention to the taste of the beverage and to interoceptive and behavioural signs of intoxication. Furthermore, it is also important that the participant be engaged in some activity that draws attention away from focussing on such interoceptive physical, or psychological stimuli (Marlatt & Roshenow, 1980).

To ensure that the manipulations had been effective in influencing the participant's expectations, most studies implement a manipulation check, usually in the form of questionnaires or interviews at the post experimental debriefing. The usual procedure involves asking participants to identify which group they were in, whether they received alcohol, or to give an estimation of their blood alcohol concentration. Most studies have revealed that those participants who expected alcohol estimated higher levels of alcohol than those who expected tonic (Knight et al., 1986).

However, these manipulation checks may be confounded by demand characteristics. When participants are asked to identify their behaviour, alcohol level, or assigned group, it is possible that they respond partially by repeating the previous instructions of the experimenter. Further inquisition regarding the alcohol content of their beverage or their level of intoxication may oblige participants to reply in a manner consistent with their previous response. Also, because it is often the same experimenter who conducted both the study and the manipulation check, it is likely that participants would find it stressful to challenge the experimenter, especially if good rapport had been established (Knight et al., 1986).

Comparatively, it is not unreasonable to suppose that the manipulation check may prompt participants to evaluate more closely the congruence between their subjective experience and their previous instructions (i.e., expectancy manipulation). If deliberate self observation did occur, it might result in an increased awareness of any discrepancy, and a self report at variance with the instructions. Again, this seems especially plausible if the questions are asked by the same person who gave the initial beverage instructions (Brick & Pohorecky, 1983).

Several studies have attempted to resolve these potential problems with experimenter demands. Brown (1981) cited in Bradlyn and Young (1983), reports that at moderate blood alcohol levels, there is no significant effect on subsequent responding when participants are asked which beverage they thought they had been given half way through the drinking period, compared with asking them at the conclusion of the experiment. Knight et al. (1986) in a study of the experimenter demands on expectancy, report that participants who believed they had been given alcohol estimated greater amounts of alcohol in their drinks than participants who believed they had consumed a non-alcoholic beverage. Actual drink content had a much smaller effect.

Despite the number of studies using the balanced placebo design to investigate the relationship between anxiety and alcohol, many of the assumptions underlying the basic components of the balanced placebo design have yet to be adequately assessed (Bradlyn & Young, 1983). For instance, Goldman et al. (1987) suggest that the balanced placebo design only provides indirect evidence for the operations of expectancies. Critchlow-Leigh (1989) notes that much balanced placebo design research assumes without empirical support, that the belief that one has consumed alcohol affects behaviour, because of individual causal beliefs about the properties of alcohol (i.e., outcome expectancies). Although the balanced placebo design has shown that instructional set affects behaviour and affective states, studies attempting to relate individual beliefs about the effects of alcohol (i.e., outcome expectancies) to behaviour have shown divergent results(Corcoran & Parker, 1991; Critchlow-Leigh, 1989; Critchlow-Leigh, 1990). The experimental manipulation is limited to instructional set about the beverage being administered, and the provision of tangible stimulus cues such as mixing drinks from labelled bottles. Further validation of the expectancy concept and its relationship to outcome expectancies requires that researchers obtain a more direct means of assessing and manipulating expectancy content itself.

ALCOHOL AND ANXIETY STUDIES USING THE BALANCED PLACEBO DESIGN

A number of studies have been published that have employed the balanced placebo design to investigate expectancy and pharmacological factors involved in alcohol consumption, and their relationship to anxiety or stress. However, as noted, the pattern of results is inconsistent, and the relationship between alcohol and anxiety remains inconclusive. The literature becomes even more inconsistent when considering the sex differences seen in the effects of alcohol expectancy. Three response systems have been identified that are presumed to reflect stress or anxiety; physiological (i.e., measurement of heart rate, galvanic skin response); behavioural (i.e., observational measurement), and self report (i.e., measured by mood checklists). Of these three response modes, self report of anxiety shows the greatest experimental controversy (de Boer, Schippers, & van der Staak, 1993). Alcohol expectancy effects, particularly with regard to sex differences are also weak or absent with self report measures and, therefore, these measures will receive the greatest attention in the following review.

In the first of the balanced placebo design studies investigating alcohol consumption and anxiety, Polivy et al. (1976) measured self report anxiety in the context of an electrical shock threat. An anxiolytic or sedative effect of alcohol consumption was seen, as men who received alcohol reported themselves to be less anxious than those who received a non-alcoholic beverage. In addition, however, alcohol expectancy resulted in an anxiogenic effect in male social drinkers. Those participants who expected alcohol gave higher ratings of anxiety than those who expected a non-alcoholic beverage.

The possibility that the belief that one has consumed alcohol may alter anxiety levels, irrespective of actual alcohol content, was later confirmed by Wilson and Abrams (1977) and Abrams and Wilson (1979). In two experiments of almost identical design, the effects of alcohol consumption on self report social anxiety, and physiological arousal in male and females, respectively, was investigated. Social anxiety, suggested to be a more meaningful anxiety manipulation than threat of electric shock (Marlatt & Rohsenow, 1980), was measured in a situation in which participants were asked to make a favourable impression on a confederate of the opposite sex, who remained neither verbally nor non-verbally responsive. Wilson and Abrams (1977) and Abrams and

Wilson (1979) demonstrated a predominant effect of alcohol expectancy on physiological measures of social anxiety, but a weaker expectancy effect with self report measures. Alcohol expectancy was found to increase anxiety in women and comparatively, to decrease anxiety in men. Alcohol consumption (i.e., pharmacological mechanisms), however, showed no significant effect on anxiety.

However, more recent studies have failed to find a significant expectancy effect on anxiety, but rather report significant and predominant effects of alcohol on anxiety levels. These studies generally suggest that alcohol consumption results in an anxiolytic or stress response dampening effect, irrespective of alcohol expectancy (Sher & Walitzer, 1986; Wilson, Brick Adler, Cocco, & Breslin, 1989). Female participants were used in the Wilson et al., (1989) study and male participants were used in the study by Sher and Walitzer (1986). Alcohol dosages in the abovementioned studies varied, with a 0.425g/kg dosage (Sher & Walitzer, 1986) and a 1:3 ratio of vodka to tonic achieving a blood alcohol concentration of 40 to 80 mg/dl (Wilson et al., 1989).

For instance, McCollam, Burish, Maisto, and Sobell (1980) report that in men, alcohol consumption (doasge = 0.64ml of 100% ethanol/kg of body weight) resulted in increases in ratings of positive affect, but that instructional set (i.e., alcohol expectancy) had little effect on self report measures of affect. Similarly, Levenson et al. (1980) found that alcohol decreased heart rate and self report anxiety in response to both shock and self disclosing speech stressors in male participants.

Abrams, Lipscomb, and Wilson (1980) report that alcohol's effects on social anxiety in male social drinkers were dose dependent, with significantly greater anxiolytic effects seen at a high dose of 1g/kg. Both measures of autonomic arousal and behavioural observations during an interaction in which they were asked to make as favourable an impression as possible on a female confederate, showed this effect. Self report anxiety did not, however. Sher and Walitzer (1986) subsequently replicated this finding of a dose dependent relationship between alcohol and heart rate reduction in male social drinkers. However, a significant anxiogenic effect of alcohol consumption was seen with a much lower dosage of alcohol (i.e., 0.425 g/kg). They also found a significant but weaker effect on self report of anxiety. There was no evidence that expectancies regarding alcohol were associated with stress responsivity in participants.

Hull and Bond (1986) provide a meta-analysis on research investigating the effects of alcohol consumption and expectancy within the balanced placebo design. This involved a review of fourteen studies examining the effects of alcohol and expectancy on various states and behaviours, including self reported mood. Self report mood was achieved by measuring either positive mood, negative mood, or both, and for the purpose of the analysis, reduction in negative mood was considered comparable to augmentation in positive mood. The meta-analysis indicated that alcohol consumption significantly enhances mood and that this effect was relatively homogenous across the fourteen studies. Alcohol expectancy was found not to affect mood when all studies were combined, yet significant heterogeneity of expectancy effects was seen, due to interactions with sex.

Hull and Bond (1986) explain this heterogeneity of expectancy effects, by an examination of differences across the experiments. The obvious difference between the fourteen studies using the balanced placebo design was the positive, negative, or neutral nature of the experimental context. For instance, several of the fourteen studies (e.g., Connors & Maisto, 1979; McCarty, Diamond, & Kaye, 1982; McCollam et al., 1980; Steele & Southwick, 1985) placed participants in relatively neutral settings, while several other studies (Abrams & Wilson, 1979; Levenson et al., 1980; Polivy et al., 1976; Rimm et al., 1981; Wilson & Abrams, 1977) exposed their participants to anxiety arousing situations. Meta-analytic results showed that expectancy has a more beneficial effect on mood in a humorous setting than in a neutral setting, and expectancy had no differential effects in anxiety arousing versus neutral settings. Hull and Bond (1986) suggest that expectancy had no effect in anxiety arousing situations because participants feel little motivation to express the emotion that they are feeling.

Sex differences in alcohol expectancy effects.

Available literature suggests that the sex differences in the effects of alcohol on anxiety relate more to expectancy effects (i.e., cognitive factors) than pharmacological mechanisms (Sher, 1987). Experimental literature shows evidence that male and female social drinkers respond differently to the belief that alcohol has been consumed.

The importance of sex in moderating the effects of alcohol on anxiety has been investigated by Wilson and Abrams (1977), Abrams and Wilson (1979) and Sutker, Allain, and Brantley (1982). Although the methods and results of these investigations were different, in all studies it appeared that expectancy for alcohol was more anxiety inducing for women than men, whereas the pharmacological effects, when present were comparable across sexes.

To clarify, Wilson and Abrams (1977) found that men who believed they had consumed alcohol were physiologically less aroused than men who believed they had consumed a non-alcoholic drink. Again, self report measures revealed a similar, though weaker expectancy effect. Comparatively, Abrams and Wilson (1979), investigating women found that women who believed that they had consumed alcohol were more anxious when compared with women who believed themselves to have consumed a non-alcoholic drink. This sex differences in the effects of alcohol expectancy on anxiety, was a particularly significant finding as alcohol research pays little attention to sex differences in the effects of alcohol (de Boer et al., 1993).

In support of Abrams and Wilson (1979), Sutker et al. (1982), also found that alcohol expectancy led to an increase in anxiety in females. Anxiety was induced prior to beverage administration by threat of a harmless, but painful electric shock for inevitable cognitive performance errors. Results showed that women who expected and received alcohol showed higher levels of self report anxiety, in contrast to women who expected tonic, but received alcohol. Alcohol expectancy did not seem to exert physiologically or psychologically relaxing effects for women who expected alcohol. However, an anxiolytic effect of alcohol consumption was seen as women in the expect tonic/given alcohol condition reported significantly less anxiety than all other groups. Neither alcohol expectancy nor consumption was associated with changes in anxiety for men. It must be noted that these results are difficult to interpret because the threat of shock as an anxiety manipulation is open to criticism, since it is unlikely that many people have developed expectancies about the effects of drinking upon an impending electric shock (Marlatt & Roshenow, 1980).

Comparatively, Levenson, Oyama, and Meek (1988) report that alcohol consumption reduced physiological measures of stress equally in both men and women. Wilson et al . (1989) utilising female drinkers, investigated the effects of different dosages of alcohol on anxiety using a stressful social interaction task. A moderate dose of alcohol resulted in increased anxiety in the social interaction tasks, whereas a high dose of alcohol decreased self report anxiety. Expectancies about intoxication failed to significantly affect anxiety in women.

A more recent study by deBoer et al. (1993) further elucidates sex differences in alcohol expectancy effects. de Boer et al. (1993) undertook a partial replication of the studies by Abrams and Wilson, but increased experimental control by assessing men and women, with similar drinking histories, in the same experimental session. Results showed an anxiolytic effect of alcohol consumption, following an anxiety provoking self disclosure task, in both men and women. Although alcohol expectancy was found not to have a main effect on anxiety levels, expectancies were involved in an interaction with sex. Alcohol expectancy was found not to influence anxiety in men, whereas women who believed they had consumed alcohol were less anxious than women who believed they had consumed tonic water. This effect for women was in the opposite direction to that found by Abrams and Wilson (1979).

Explaining sex differences

This sex difference, seen predominantly in the effects of alcohol expectancy on anxiety may be explained in a number of fashions. de Boer, Schipper, and van Der Staak. (1994) have recently suggested that men and women respond differently to the belief that alcohol has been consumed, because of differing direct and indirect experiences and histories with alcohol, resulting in variance in the content of alcohol related beliefs (i.e., outcome expectancies). In support of this, studies investigating the content of alcohol related beliefs have shown that individual beliefs may vary dependent upon the individual's alcohol consumption and drinking history and culture (Goldman, et al., 1991).

According to social learning theory (Abrams & Niaura, 1987) alcohol related beliefs may interact with the pharmacological effects of alcohol and determine in part, intoxication experienced changes in affect. In terms of a sex bound social learning history alcohol is believed to cause disinhibition of sexual and aggressive responses, while at the same time reducing anxiety (Critchlow- Leigh, 1990). This belief may have a different impact on men and women. Whereas for men, losing control after alcohol consumption has a positive connotation, women who drink and lose control are socially sanctioned.

In support of this, Marlatt and Roshenow (1980) note that divergent empirical findings for males and females may reflect the ambivalent attitudes that society holds about women who drink and the effects of alcohol on their behaviour. Although society seems to accept that drinking is an acceptable stimulus for "masculine" behaviours (e.g., aggressive and sexual acting out), it is far more critical about acceptable reasons for women to drink. It would seem that the general consensus is that women who drink in social situations, have more to lose if their self control and inhibitory processes are diminished. Consequently, women may have become fearful of alcohol induced disinhibition, and may experience anxiety when instructed that they will be consuming alcohol.

This idea is supported by research on alcohol related beliefs, which has shown that women report overall lower positive expectancies, less specific expectancies, and higher expectancies of alcohol's negative effects than men (Gustafson, 1991). Rosenhow

(1983) reports that women generally express expectations of less relaxation and tension reduction from a few drinks than men.

Contradictory results seen in the studies of Abrams and Wilson (1979) and de Boer et al. (1994), in which alcohol expectancy increased and decreased anxiety in women respectively, may also be explained in terms of alcohol related beliefs. It is salient to note that the studies of Abrams and Wilson (1979) and Wilson and Abrams (1977) may have been confounded by an absence of adequate control for drinking history. Male and female participants in these studies were not matched for drinking history and furthermore, were not studied in the same experimental situation. For instance, women reported shorter histories of experiences with alcohol and also less consumption of alcohol per week, than their male counterparts. Perhaps if the women in Abrams and Wilsons' (1979) study had more drinking experience they might have responded more favourably to the expectation of alcohol. Furthermore, the studies of Wilson and Abrams (1977) and Abrams and Wilson (1979) were conducted over nearly two decades ago. Sex role stereotypes may have changed and the double standard concerning sexual behaviour suggesting that women learn to be more wary and suspicious when drinking may have lost its impact as women increasingly tend to adopt male drinking patterns (de Boer et al., 1993). Perhaps the opposite sex effect seen by de Boer et al. (1993) in which alcohol expectancy decreased anxiety in women, may be explained by the presence of more positive alcohol beliefs in their participants. This is likely, as contemporary society has fewer and less extreme social sanctions for women regarding drinking (de Boer et al., 1994).

Thus, differences in social learning history, and/or cognitive factors could account for differences in results between males and females in the relationship between alcohol expectancy and anxiety. Consequently, not sex per se, but beliefs that differ between sex may be an important determinant of the variety of effects that alcohol may produce on anxiety (Wilson, 1982; de Boer et al., 1994)

Issues of measurement and meaning

The equivocal nature of the relationship between alcohol and anxiety may be partially attributed to the inadequacy and heterogeneity of methods and measures used to investigate the effect of alcohol on anxiety. Different studies have used different methodologies, making comparison across studies extremely difficult, if not impossible. Some of these methodological differences include; alcohol administration, consumption and absorption (i.e., stomach contents, type of beverage, and dosage of alcohol); temporal factors regarding assessment of dependent variables (i.e., time of day, phase of blood alcohol function); drinking environment, physical and social setting, and type of participant (i.e., sex, prior drinking history, tolerance) (Connors & Maisto, 1983).

Another issue that permeates alcohol and anxiety research is the definition and measurement of the "anxiety construct" itself. There are no definitive statements that can be made about the phenomenon labelled "anxiety". The global terms "anxiety", "tension" and "stress" are used synonymously, since attempts to draw finer distinctions have typically been either too global or too circumscribed to have utility (Wilson, 1988). Thus, the term has different meanings for different people, and consequently, remains a convenience for communication, with limited scientific value (Connors & Maisto, 1983). A major problem with this issue of "meaning" is that the way stress is operationalised may restrict the generalisations that can be made between the laboratory and the natural environment.

Problems in developing an acceptable definition of anxiety have led researchers to specify measurement procedures and to identify the parameters of the various response systems presumed to reflect stress. Influenced by Lang (1969) and Rachman's (1978) triple response system analyses of anxiety, three general response categories have been identified. These domains are; physiological (i.e., autonomic nervous system arousal-heart rate, muscle tension etc.); motoric (i.e., overt behavioural expressions), and self report (i.e., self referent statements/ responses to mood adjective checklists) (Connors & Maisto, 1983). Anxiety has since been assessed in congruent fashions, including diverse forms of self report, different measures of autonomic nervous system arousal, biochemical indices, and observational and behavioural measures (Wilson, 1988).

Although, stress has been inferred from measurement of these response systems, it is unclear how different response modes should be combined, or at what threshold value stress or anxiety is accepted to exist (Abrams, 1983). Furthermore, these different response systems do not always change in the same direction, producing a desynchrony which further complicates interpretations of alcohol and anxiety research. It is possible that the different factors that influence anxiety have partially separate effects on the different response systems that comprise anxiety. Researchers need to concentrate on identifying and validating indices of anxiety or stress and the patterning of their covariance and therefore it is necessary to determine the inter-relationships among different response modalities and to integrate them (Connors & Maisto, 1983).

Unfortunately, anxiety induction procedures have also varied widely including; experimentally induced conflict and aversive stimuli, (e.g., fear of electric shock) (Polivy et al., 1976); social stressors / performance anxiety (e.g., the anticipation of self disclosing or public speaking or stressful social interaction)(de Boer et al., 1993); and phobic avoidance. Similarly, the perceived intensity and the extent to which laboratory stressors significantly strain a participant's self regulation capacity have not received sufficient attention (Abrams, 1983).

Also rarely addressed is the temporal patterning of intoxication and exposure to the stress induction procedure. Most alcohol and anxiety research has adopted a procedure in which participants are first intoxicated and then exposed to the stressor (Wilson, 1988). This procedure may ignore the importance of emotional state before alcohol consumption, and possibly is better suited to testing the notion that alcohol intoxication reduces tension susceptibility, rather than pre-existing tension or negative states.

Research using a reversed order design, in which participants are exposed to the stressor before intoxication has failed to show anxiogenic effects (Sutker et al., 1982). For instance, Sayette, Wilson, and Carpenter (1989) found that participants who received a stressor before intoxication experienced a greater stress response than participants who received the stressor after intoxication. This is inconsistent with above-mentioned research in which the stressor followed intoxication resulting in an anxiolytic effect on stress (Wilson, 1988).

Considering the above difficulties in measurement and meaning, it is not surprising to find equivocal results in the literature. Various researchers (e.g., Hull &

Bond, 1986) have called for more careful attention to the selection of appropriate stress induction procedures, greater comparability across studies and for triple response mode assessments when conducting research on alcohol and anxiety relationships.

CONCLUSION

The available empirical research has revealed that there is no simple relationship between alcohol and its effect on anxiety. Rather, it must be stated that the effects of alcohol on anxiety are a complex function of both pharmacological, cognitive and situational factors. In fact, Wilson (1988) states that "asking the question 'does alcohol reduce anxiety' is conceptually naive." This often made assumption of some invariant and automatic effect of alcohol on anxiety ignores the multiple bio-psycho-social factors of which this effect is a function. Wilson (1982) suggests that the more meaningful question that must be addressed is "At what dose, under what conditions, in whom and on what measures does alcohol reduce anxiety?"

Further research must address the methodological concerns mentioned above in order to gain substantiative and reliable results about the effects of alcohol consumption and expectancy on anxiety. Future research would benefit from assessing male and female participants, with similar drinking histories in the same experimental situation. Similarly, triple response mode assessments of anxiety should be more frequently utilised and the meaning of anxiety more clearly defined. Finally, more attention to the assessment of self report anxiety would seem warranted because of the illustrated inconsistency in self report of anxiety.

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Alcohol and anxiety: The influence of pharmacological mechanisms, alcohol expectancy, and sex.

Abstract

The present study was conducted to determine the influence of pharmacological and expectancy mechanisms on simple reaction time and self report anxiety of men and women in a task designed to provoke performance anxiety. Ten male and ten female social drinkers participated in a 2 (sex) x 2 (alcohol expectancy) x 2 (beverage content) factorial balanced placebo study. The between subjects design generally used in balanced placebo research was replaced by a within subjects design, involving each participant completing four experimental sessions. Although results showed no significant effect of alcohol consumption, alcohol expectancy, or sex on self report anxiety, a number of trends were evident for self report anxiety. Firstly there was a trend suggesting that overall women were more anxious than men. Secondly an interaction between pre/post anxiety and instructional manipulation for both male and female anxiety responses approached significance. People who were told that they were receiving alcohol were more anxious following drink consumption than before drink consumption regardless of whether there was any alcohol in their beverage or not. No significant main effects for alcohol consumption, expectancy or sex were seen for reaction times to hits or false alarms, or to numbers of hits and misses.

The present study is concerned with the relationship between alcohol consumption and anxiety in men and women. Particular interest is directed toward the influence of pharmacological mechanisms and cognitive expectancy factors.

It is a commonly held belief that, under the influence of alcohol, people experience a reduction in anxiety and become more outgoing, friendly, and relaxed. This intuitive relationship between alcohol and anxiety has been the focus of much empirical research. Despite this, the relationship between alcohol consumption and anxiety is neither well characterised nor well understood. Alcohol consumption has variously been demonstrated to reduce anxiety, to increase anxiety, and to have a neutral effect (Young, Oei, & Knight, 1990).

Most early research assumed that alcohol influenced behaviour and anxiety levels because of its pharmacological impact upon perceptual, cognitive, and/or motivational states (Hull, 1981). Alcohol, a central nervous system (CNS) depressant, potentiates the action of the gamma amino butyric acid (GABAb) receptor, the major inhibitory neurotransmitter in the brain. This enhanced GABA transmission results in relaxation and decreased anxiety levels (Kalat, 1995; Carlson, 1994).

However, since the 1970's attention has been directed to the influence of cognitive processes or expectancy factors in the mediation of the effects of alcohol (Bandura, 1985). In empirical research, using the balanced placebo design, alcohol expectancy refers to the participant's belief that he or she has received an alcohol beverage (Crichlow-Leigh, 1989). Alcohol expectancy or the belief that one has consumed alcohol, irrespective of actual drink content, has been suggested to be sufficient to lead to disinhibition of behaviour or alteration of affective states (Hull & Bond, 1986).

Alcohol expectancy may be differentiated from the beliefs that people hold about the effects of alcohol, known as outcome expectancies or alcohol related beliefs. The nature of the relationship between alcohol expectancy and outcome expectancies is uncertain, although researchers generally accept that alcohol expectancy effects are directly linked to, and, are mediated by the content of outcome expectancies (Stacy, Newcomb, & Bentler, 1991)

Incorporating the importance of both pharmacological mechanisms and expectancy effects is a central feature of the social learning theory of alcohol use (Abrams & Niaura, 1987). The theory broadly suggests that alcohol related beliefs (i.e., outcome expectancies) may interact with the pharmacological mechanisms of alcohol to determine, in part, intoxication experiences. The model also provides explanations for the development of alcohol related beliefs and expectancies, and consequently, how the content of these beliefs may mediate either anxiolytic or anxiogenic effects in people. Similarly, through an examination of social norms and sanctions regarding appropriate drinking behaviour for men and women, the model provides an adequate explanation for sex differences in the effects of alcohol expectancy on anxiety.

According to this model, beliefs, attitudes, and expectancies concerning alcohol are formed vicariously through socialisation very early in life, prior to actual experiences with alcohol, through the influence of family, media and peers. From an early age individuals learn how alcohol is used, and in what situations, and what behaviours are permitted for each sex, when intoxicated (MacAndrews & Edgerton, 1969).

Direct experience with alcohol later in life is believed to crystallise these vicariously learnt beliefs, attitudes, and expectancies. (Christiansen, Goldman, & Brown, 1985) For example, when tension reduction, mood enhancement, or better social interaction is experienced while drinking, this direct experience reinforces the belief that alcohol helps in social interaction situations.

As aforementioned, individual beliefs are believed to mediate expectancy effects of alcohol (Young et al., 1990) and therefore, differences in alcohol related beliefs may explain the inconsistency in expectancy effects on anxiety. For instance, if one holds negative beliefs about alcohol, due to aversive past experiences, alcohol related beliefs, and expectancies, alcohol expectancy in a laboratory situation is likely to have an anxiogenic effect. Conversely, if positive alcohol related beliefs are held, then alcohol consumption is more likely to have an anxiolytic effect.

By means of a balanced placebo design, in which beverage content and expected beverage content are factorially crossed, the effects of cognitive expectancy and pharmacological mechanisms on anxiety levels have been independently assessed. Findings have been inconclusive, and both pharmacological factors and cognitive expectancy have been purported to be primary factors influencing anxiety levels following alcohol intoxication. Similarly, the direction of the influence (i.e., increase or decrease in anxiety) is also an area of controversy and has been complicated by recent findings of a sex difference in the effect of alcohol expectancy on anxiety. This sex difference was an important finding as alcohol research shows an absence of studies using female participants (Levenson, Oyama, & Meek, 1987)

Early research utilising a balanced placebo design by Wilson and Abrams (1977) and Abrams and Wilson (1979) indicated the significance of alcohol expectancy, but not pharmacological effects in physiological, behavioural, and self report measures of anxiety in women and men, respectively. Moreover, it was found that men and women seem to respond differently to the expectancy that alcohol has been consumed. In a social interaction situation with a confederate of the opposite sex, alcohol expectancies were found to reduce anxiety in male subjects (Wilson & Abrams, 1977) and comparatively, to increase anxiety in female subjects (Abrams & Wilson, 1979). Sutker, Allain, Brantley, and Randall (1982) also report an anxiogenic effect of alcohol expectancy in their female participants.

Other studies, however report the predominant significance of pharmacological mechanisms. These studies suggest that alcohol consumption reduces anxiety, irrespective of alcohol expectancy. For instance, Sutker et al. (1982) and Polivy, Schueneman, and Carlson (1976) found an anxiety reducing effect of alcohol consumption in men and women respectively. Hull and Bond (1986) in a meta-analysis of studies utilising the balanced placebo design to investigate the effects of alcohol consumption on various social behaviours, report that alcohol enhances mood and positive affect and reduces anxiety, whereas alcohol expectancies have no overall effect in anxiety arousing situations.

More recently, in an attempt to clarify inconsistency in the literature, de Boer, Schippers, and van der Staak (1993) undertook a partial replication of the studies by Abrams and Wilson and increased experimental control by assessing men and women, with similar drinking histories, in the same experimental session. A factorial design with sex, alcohol, and expectancy as factors was utilised, with social anxiety assessed by self report measures. Results showed an anxiety reducing effect of alcohol consumption, following an anxiety provoking self disclosure task. Although alcohol expectancy was found not to have a main effect on anxiety levels, expectancies were involved in an interaction with sex. Expectancies were found not to influence anxiety in male subjects, whereas women who believed they had consumed alcohol were less anxious than women who believed they had consumed a non-alcoholic beverage.

This effect for women was in the opposite direction to that found by Abrams and Wilson (1979). This discrepancy may have been the result of experimental inadequacy. Firstly, the male and female participants in the studies by Wilson and his colleagues were not studied in the same experimental situation. Secondly, the female participants in Abrams and Wilson (1979) study had shorter drinking histories and consumed less alcohol per week than male participants in the Wilson & Abrams' (1977) study. Comparatively, the male and female participants in the research by de Boer et al. (1993) were matched for drinking history and studied in the same experimental situation and as such more faith may be laid in the results of de Boer et al. (1993).

Finally, the pharmacological and behavioural effects of alcohol consumption can be shown in complex reaction time performance. While research has generally shown that increases in blood alcohol concentration are associated with increased reaction times (Maylor, Rabbitt, James, & Kerr, 1992), this is not a conclusive finding. For instance, Krull, Smith, and Parsons (1994) report that a moderate dose of alcohol slowed reaction time in their study, whereas, Finnigan, Hammersley, and Miller (1995) report that a high dosage of alcohol caused a significant slowing of reaction time that was not seen with moderate or low doses of alcohol. Comparatively, expectancy effects are decreased or absent altogether with reaction time tasks (Marlatt & Rohsenow, 1980), although Ross and Pihl (1988) report a significant effect of alcohol expectation on reaction time. Participants in this study who were told that they received a high dosage of alcohol had faster reaction times that those participants told that they received a lower dosage of alcohol. The present study will utilise a complex reaction time task as a measure of the behavioural effects of alcohol consumption.

Hence, the present study is undertaken with the aim of clarifying the relative and differential influences of cognitive expectancy and pharmacological mechanisms (i.e., alcohol consumption) on anxiety levels in men and women. Prior research has been limited in a number of ways. Firstly, there has been an absence of research utilising female participants and similarly, research comparing two sexes, with similar drinking histories in the same study is rare. The present study will enhance experimental literature by studying both males and females, with similar drinking histories (i.e., social drinkers) in the same experimental situation.

Secondly, prior research applying the balanced placebo methodology has utilised a between subjects design in which participants are randomly assigned to each of the four experimental conditions. The present study departs from this methodology by utilising a within subjects design, for a number of reasons. Firstly, to examine the utility and validity of the BPD using a within subjects design and, secondly to enhance experimental control by allowing each participant to act as his/her own control

Several researchers have also called for triple mode assessment (i.e, behavioural, self report, and physiological) of anxiety (Hull & Bond, 1986) and several others have noted that the effects of alcohol expectancy on self report anxiety is an area of inconsistency in alcohol and anxiety literature. In consequence, it was decided to use, a behavioural measure of performance (i.e., reaction time) and a self report measure of anxiety.

A number of predictions will be addressed in this research. Prior research has shown that alcohol consumption generally results in anxiety reduction (Sutker et al., 1982; Hull & Bond, 1986; Polivy et al., 1976), and also that alcohol consumption generally slows reaction time (Maylor et al., 1992; Krull et al., 1994). In support of this, the first prediction is that alcohol consumption will cause a significant decrement in reaction time and the second prediction is that alcohol consumption will result in an anxiogenic effect for both men and women.

In keeping with Marlatt and Roshenow (1980), who report that alcohol expectancy has little effect on reaction time performance, the third prediction is that alcohol expectancy, will have a limited or absent effect on reaction time. Prior research has also shown evidence of sex differences in the effects of alcohol expectancy on social and performance anxiety (de Boer et al., 1993; Wilson & Abrams, 1977; Abrams & Wilson, 1979). The final prediction of the present study is that alcohol expectancy will significantly affect self report anxiety and that this will be involved with an interaction with sex.

METHOD

Participants

Participants were Psychology One students who volunteered in order to complete undergraduate course criteria. Twenty eight participants (14 males and 14 females), who were moderate social drinkers (i.e., 3-5 drinks in one session) and aged between 18 and 40 years participated. Participants were asked to complete four sessions (duration = one and a half hours), approximately one week apart. Eight of these participants were excluded for data analysis due to unsatisfactory participant commitment and incomplete data collection. Exclusion parameters for potential participants included: major health problems; the use of prescribed drugs; a history of drug or alcohol abuse; head injury or brain damage; epilepsy; poor vision; pregnancy; and either absence of a driving license, or driving experience that was less than two years.

Design

A (2) x 2 x 2 mixed design was used. The between groups factor was *Sex* (male; female) and the within groups factors were *Beverage Content* (given alcohol; given no alcohol) and *Alcohol Expectancy* (told alcohol; told no alcohol).

Hence, each participant completed the following four experimental sessions, scheduled, approximately one week apart. The order of these conditions was counterbalanced across all participants.

1. Told alcohol/given alcohol
2. Told alcohol/given no alcohol
3. Told no alcohol/given alcohol
4. Told no alcohol/given no alcohol

The dependent variables were reaction time to hits and false alarms, numbers of correct rejections, false alarms and misses and responses on the State portion of the State

Trait Anxiety Inventory (STAI: Spielberger, Gorusch, & Luscherne, 1970- See Appendix A).

Drink and expectancy administration

1. Given alcohol conditions: In female participants, a 0.95 ml/kg of body weight dose of alcohol, calculated to give rise to a blood alcohol concentration of 0.05 % was measured. The same formula was used for males, but with a 15 % increase in quantities of orange juice and alcohol. Peppermint water was added in the ratio of 0.1 ml/kg of body weight to provide a mask for the alcohol and placebo conditions. Orange juice was then added in the ratio of 5.7 ml/kg of body weight.

2. *Given no alcohol conditions*: The same quantity of orange juice(as in 1.) and peppermint water was combined.

3. Told alcohol conditions: Orange juice and real or apparent vodka (i.e. water in a vodka bottle) was mixed in the participants' presence, according to the appropriate alcohol expectancy manipulation.

4. Told no alcohol conditions: Orange juice or a premixed combination of vodka and orange juice were poured from an orange juice container, according to the appropriate alcohol expectancy manipulation.

Simple Reaction Time Task

Participants were asked to view a series of driving scenes, presented on slides. An oddball paradigm was utilised with the probability of a rare event (i.e., accident scene) being 15%. Stimulus duration was 250 ms and the inter stimulus interval was 1100ms. Participants were required to respond as quickly and accurately as possible whenever they would normally brake in a car driving at 60km/h in response to an imminent accident scene. Data collected was reaction times to hits and false alarms and also numbers of correct rejections, hits, false alarms and misses. The collection of data stopped when 40 hits were achieved.

Anxiety Inducing Task

Participants were asked to give a three minute impromptu speech on a provided topic, in front of a non-functional video camera which they were informed was working. The participants were told that this speech would be rated for quality and content when viewed later by the experimenter. The four speech questions were counterbalanced, with each participant receiving a different speech topic each session.

The four questions were-

1. What are the levels of the criminal justice system in Australia?
2. What are the different ways a building may be heated and which is the most energy efficient and why?
3. What are the major tourist attractions in Australia?
4. What are the five major food groups and what functions do they serve in our bodies?

Measurement of Anxiety

Self report anxiety was measured immediately after the participant received his/her beverage and also at the completion of the anxiety inducing task. Anxiety was measured using the State portion of the State -Trait Anxiety Inventory (Spielberger et al., 1970).

Procedure

Potential participants were screened using a medical history and drinking questionnaire (see Appendix B) and were selected according to the above-mentioned exclusion criteria. Participants were give information sheets, signed informed consent forms and were asked to abstain from alcohol for twenty four hours, and from food for four hours before each experimental session. The duration of each session was approximately one and a half hours and the interval between the four sessions, approximately one week.

At the beginning of each session, participant blood alcohol concentration (to ensure a 0.00 blood alcohol concentration) and blood pressure were measured. Following this, each participant was informed that they would receive either alcohol, orange juice and peppermint water (Told alcohol/given no alcohol and the told alcohol/given alcohol conditions) or just orange juice and peppermint water (told no alcohol/given no alcohol and told no alcohol/given alcohol conditions). Drinks were mixed and administered in the abovementioned fashion and each participant ingested the drinks over a period of twenty minutes.

Thirty minutes after the ingestion of the beverage, the blood alcohol concentration of each participant was measured several times in each of the four conditions. This was done to ensure that the participant did not become suspicious when blood alcohol concentration readings were taken in the told no alcohol/given no alcohol condition. The readings on the breathanalyser were not shown to the participants at any time. In the told alcohol/given no alcohol conditions, the participant was told that he/she had a blood alcohol concentration of 0.05 and in the told alcohol/given alcohol and in the told no alcohol/given no alcohol conditions, blood alcohol concentration was measured until levelsblood alcohol concentration peaked.

Following this, the reaction time task and the anxiety inducing task were respectively completed. At the end of each experimental session, each participant completed a subjective intoxication rating scale and also estimated their blood alcohol concentration. Unfortunately, the data for the subjective intoxication rating and blood alcohol estimation was destroyed due to flooding. Following completion of the final session, participants were debriefed regarding the nature of the experiment and the deception involved.

RESULTS

Data collection involved scoring participants' responses on the State form of the State-Trait Anxiety Inventory (Speilberger et al., 1970) completed before and after the anxiety provoking task for degrees of self report anxiety. The reaction time task that participants undertook in each session provided reaction times to hits and false alarms, and also numbers of correct rejections, misses, and false alarms. Data analysis involved a series of (2) (sex) x 2 (beverage content) x 2 (expectancy manipulation) between groups, repeated measures ANOVAs on each of these dependent variables, followed by Newmans Keuls post hoc tests where necessary. Raw data and analyses are provided in Appendix C.

Self Report Anxiety

Table 1 summarises self report anxiety for each of the experimental conditions. As illustrated in Table 1 the highest anxiety mean was seen for post anxiety responses of women in the told no alcohol/received alcohol condition (37.3). The lowest anxiety mean

(28.0) was seen in anxiety responses taken before the anxiety provoking task for men in the told no alcohol/given no alcohol and told no alcohol/given alcohol conditions.

Table 1. Means of anxiety responses for all conditions

	Males				Females			
	pre	**Std.Dev.**	**post**	**Std.Dev.**	**pre**	**Std.Dev.**	**post**	**Std Dev.**
told alcohol/ given alcohol	29.5	5.72	30.1	9.68	33.4	7.2457	34.9	14.34
told no alcohol/ given alcohol	33.8	5.52	30.5	7.48	33.8	10.40	37.3	8.25
told alcohol/ given no alcohol	28.0	4.98	31.8	6.54	30.8	7.48	34.6	8.43
told no alcohol/ given no alcohol	28.0	6.74	29.0	6.05	31.1	6.35	36.8	11.14

The four way, between groups, repeated measures ANOVA , 2 (sex) x 2 (beverage content) x 2 (instructional manipulation) x 2 (Pre/Post anxiety)] completed on State Anxiety responses revealed no significant main effects for any of the independent variables. However, there was a trend was towards significance for sex ($F_{(1,19)} = 3.30$, $p = 0.087$). Overall, females (mean = 34.19) tended to be more anxious than males (mean = 30.10).

No significant interactions between the independent variables were evident on State Anxiety scores. The interaction between pre and post anxiety and instructional manipulation (told alcohol/told no alcohol) approached significance ($F_{(1,18)} = 3.20$, $p = 0.092$). This trend is shown in Figure 1. Newman Keuls post hoc tests showed that all people who were told that they were receiving alcohol were more anxious following drink consumption than before drink consumption ($p = 0.009$), regardless of whether there was any alcohol in their beverage or not.

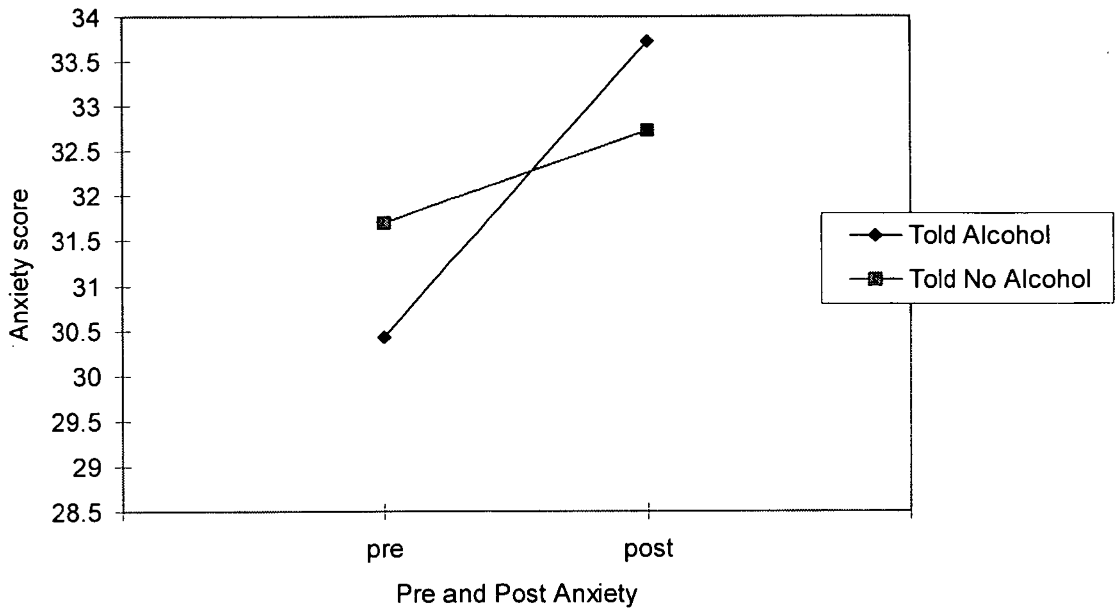

Figure 1. State-Anxiety responses across pre and post anxiety, and instructional manipulation.

Reaction times for hits and false alarms

Table 2 shows mean reaction times for hits and false alarms for all conditions. As illustrated in Table 2 the slowest mean reaction time for hits (647.2 ms) was for males in the told alcohol/given alcohol condition, and the fastest mean reaction time was for females in the told alcohol/given no alcohol condition (629.9 ms). The slowest mean reaction time for false alarms (663.00 ms) was for males in the told alcohol/ given alcohol condition and in the told no alcohol/given alcohol conditions. The fastest mean reaction time for false alarms (591.00 ms) was for females in the told alcohol/given no alcohol condition.

Table 2 Mean reaction times (ms) for hits and false alarms

	Males				Females			
	Hits	**Std.Dev.**	**False Alarms**	**Std.Dev.**	**Hits**	**Std.Dev.**	**False Alarms**	**Std.Dev.**
Given alcohol /told alcohol	647.2	55.06	663.2	116.44	629.9	73.50	647.8	86.64
Given alcohol /told no alcohol	630.6	77.3	642.3	116.16	631.7	79.3	636.3	72.32
Given no alcohol/told alcohol	635.9	51.20	663.0	116.77	621.7	75.59	591.2	61.5
Given no alcohol /told no alcohol	625.9	58.01	615.00	80.27	633.4	73.50	641.4	100.71

A three way, 2 (sex) x 2 (beverage content) x 2 (instructional manipulation) between groups, repeated measures ANOVA showed no significant main effects or interactions for any of the independent variables on reaction times for hits. A similar three way, between groups, repeated measures ANOVA showed no main effects for reaction times to false alarms. However, the three way interaction between instructional manipulation, sex, and beverage content, approached significance ($F_{(1,19)} = 3.70$, $p = 0.072$). Post hoc tests indicated that females who were told that they were receiving alcohol, but who received a non-alcoholic drink tended to have significantly faster reaction times to false alarms than males in the same condition, after beverage administration ($p = 0.07$).

Numbers of false alarms, hits, misses, and correct rejections

The largest mean for misses (10.6) was for females in the told no alcohol/given no alcohol condition and the lowest mean for misses (1.6) was for females in the told alcohol/given no alcohol condition, and for men in the told no alcohol/given no alcohol condition. The three way, between groups repeated measures ANOVA showed no significant main effects or interactions for any of the independent variables for number of misses.

The largest mean number of false alarms (10.8) was for females in the told no alcohol/given no alcohol condition and the lowest mean for number of false alarms (3.3) was for females in the told no alcohol/given alcohol condition. The three way, between groups, repeated measures ANOVA show no significant main effects or interactions for any of the independent variables in numbers of false alarms.

The largest (186) and smallest (153) mean for correct rejections was respectively for females in the told no alcohol/given no alcohol condition and for males in the told no alcohol/given no alcohol condition. A three way between groups, repeated measures, ANOVA conducted on numbers of correct rejections showed a significant two way interaction between sex and instructional manipulation ($F_{(1,19)} = 4.73$, $p = 0.04$), which is illustrated in Figure 2.

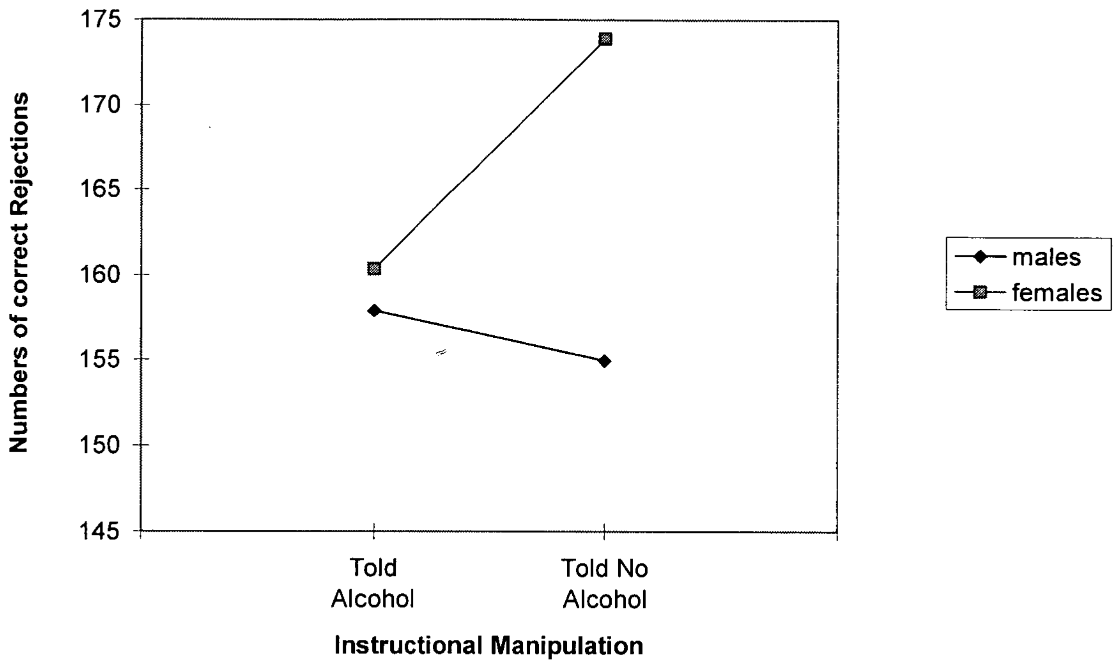

Figure 2. The interaction between sex and instructional manipulation for numbers of correct rejections.

Post hoc Newman Keuls tests indicated that females who were told that they were receiving no alcohol showed significantly larger numbers of correct rejections than females who were told that they were receiving alcohol (p= 0.02). Females who were told that they were receiving a non-alcoholic beverage showed significantly larger numbers of correct rejections than males who were told that were receiving a non-alcoholic beverage (p=0.01)

Blood alcohol Concentration

The two conditions in which participants received alcohol were in the told alcohol/given alcohol condition and in the told no alcohol/given alcohol condition. The mean BAC for females in the told alcohol/given alcohol condition was 0.033 and for males in the told alcohol/given alcohol condition the mean BAC was 0.03. For females in the told no alcohol/given alcohol condition, the mean BAC was 0.032 and for males in the told no alcohol/given alcohol, the mean BAC was 0.029.

<u>Practise effects</u>

Two separate four way, 2(sex) x 2 (beverage content) x 2 (instructional manipulation) x 4(session number), between groups, repeated measures ANOVA's showed no significant effect of session number on reaction time or anxiety responses.

DISCUSSION

The results of the present study do not provide support for prior research investigating the effects of alcohol expectancy and consumption on anxiety, and provide only partial support for the predictions of the present study. The second prediction of the present study was not supported as alcohol consumption did not significantly reduce anxiety levels in men or women. This stands in contrast to de Boer et al. (1993) and Hull and Bond (1986) who have found that alcohol consumption reduces social anxiety and increases positive mood and affect. Comparatively, the absence of a significant effect of alcohol consumption on anxiety levels may support prior research suggesting that a relatively large dose of alcohol (i.e., approximately 1g/kg-larger than given in the present study approximately) is required to demonstrate stress response dampening effects (Sher & Levenson, 1982).

Moreover, the fourth prediction of the present study was not supported because no significant alcohol expectancy effects were seen for anxiety responses, nor was alcohol expectancy involved with an interaction with sex for anxiety responses. However a number of trends were evident for anxiety responses. An interaction between pre and post anxiety and instructional manipulation (told alcohol/told no alcohol) approached significance. All people who were told that they were receiving alcohol were more anxious following drink consumption than before drink consumption, regardless of whether there was any alcohol in their beverage or not. This effect did not interact with sex.

This trend for an expectancy effect on anxiety challenges the results of Hull and Bond's (1986) meta - analysis, which showed that alcohol expectancy has no effect in anxiety arousing situations. Furthermore, the near significance of the expectancy effect on anxiety supports prior literature suggesting that expectancy effects are generally weak when the dependent variable involves self report of mood states (Marlatt & Rohsenow, 1980). Expectancy effects on anxiety have been predominantly evident in physiological and behavioural measures of anxiety, with self report of anxiety generally showing a much weaker effect (de Boer et al., 1993). Perhaps the trend for an expectancy effect on anxiety may have reached significance in the present study had physiological indices of anxiety been used, or more participants utilised (de Boer et al., 1993).

That the present trend for expectancy effects on anxiety did not differentiate between the sexes contradicts the results of Abrams and Wilson (1979) and Wilson and Abrams (1977) who report that expectancy increased anxiety in women and decreased anxiety in men, respectively. These results may be interpreted in terms of the social learning theory of alcohol use (Abrams & Niaura, 1987). The lack of a sex difference suggests that traditional sex role stereotypes regarding drinking alcohol may have changed. The traditional double standard between sexes, concerning differing appropriate drinking behaviour for men and women has suggested that women learn to be more wary and suspicious when drinking because of social sanctions. Present results suggest that this double standard may have lost some of its impact and that, perhaps, women are increasingly adopting male drinking patterns (de Boer et al., 1993) and possibly similar alcohol related beliefs. This is likely, as contemporary society has fewer and less extreme social sanctions for women regarding drinking (de Boer et al., 1994).

In a similar fashion to the results reported for anxiety measures, neither alcohol consumption nor expectancy significantly influenced reaction times to hits and false alarms. No support for the first prediction of the present study was provided as alcohol consumption did not significantly affect reaction time. This stands in contrast to prior research that has indicated significant decrement in reaction time performance at a BAC of 0.05 (Krull et al., 1994; Maylor et al., 1992). However, present results also indicated that the dosage of alcohol provided to both females (i.e., 0.95 ml/kg of body weight) and males (i.e., 0.95 ml/kg of body weight + 15%) failed to produce a BAC of 0.05. Perhaps, if a larger dosage of alcohol had been used, decrements in reaction time performance may have been seen.

Comparatively, support for the third prediction of the present study was provided as no effect of alcohol expectancy was seen for reaction time. This supports prior literature suggesting that expectancy effects are usually decreased or absent altogether with reaction time tasks (Marlatt & Rohsenow, 1980). Results of the present study must be treated with caution, however, because no effect of alcohol consumption was seen on reaction time.

It is necessary to discuss salient limitations of the present study. A number of problems became evident during the experimental testing with regard to a using a within subjects design for a balanced placebo methodology. It became clear that several of the critical assumptions of the balanced placebo design were not met.

Unfortunately, due to financial constraints and lack of resources the present study was unable to follow the double blind procedure suggested by Marlatt and Rohsenow (1980), in which a confederate is assigned the task of allocating participants to, and instructing them in, the various conditions. The experimenter in the present study knew the true nature of the beverage consumed in each condition and for each participant. Subsequently, factors such as experimenter bias and experimenter demand represent uncontrolled variables which may have confounded results. Furthermore, the results of this study may have been confounded by the effect of the experimenter's sex (female) on opposite sex (male) participants. The effects of this heterosexual interaction were not controlled for in the design.

A second issue is the credibility of the deception involved in the instructional manipulations. This issue has been a problem in several other balanced placebo designs studying alcohol and anxiety (e.g., de Boer et al., 1993). de Boer, Schippers, and van der Staak (1994) assert that there it is impossible to conduct a balanced placebo study in which all of the participants in the told no alcohol/given alcohol condition believe the deception.

In the present study, several participants in the told no alcohol/given alcohol condition indicated their suspicion about the truthful nature of their drink, by either directly asking if they had in fact been given alcohol, or by reporting internal physiological sensations associated with alcohol intake (i.e., dizziness, flushing). Furthermore, the self report measures of anxiety utilised in the present study required participants to introcept and report on subjective mood states. By directly and consciously focussing on these interoceptive cues, participants may have become suspicious about actual contents of the drinks received, which may have affected the instructional manipulation (Marlatt & Rohsenow, 1980).

The inadequacy of the instructional deception was further exacerbated by queries regarding the requirement that no participant was allowed to drive home from any experimental session. All participants were informed that they would be receiving alcohol or just orange juice at the beginning of each session, appropriate to the predetermined condition. Having knowledge of this, some participants became dubious and questioned why they could not drive home in the told no alcohol/given no alcohol and told no alcohol/given alcohol conditions.

Finally, the validity of self reports of intoxicated participants may differ from the validity of sober participants. Alcohol intoxication might interact with processes such as self report biases, sensitivity to demand characteristics, the ability to introspect and possibly even the ability to scale experience in the way required by many of the Likert like scales used in contemporary research. This issue of the differential validity of drunken and sober self reports is one that requires further attention in future experimental research regarding alcohol and anxiety (Sher, 1987)

In conclusion, it appears that using a within subjects design, for a balanced placebo methodology presents the experimenter with several problems. Questions arise regarding how to effectively induce and maintain the expectancy manipulation and also how to effectively induce anxiety. However results from the present study provide support for prior research showing that alcohol expectancy can affect anxiety levels and that it has little effect on cognitive tasks such as reaction time tasks. Perhaps if more participants completed the present study the trend that alcohol expectancy tends to increase anxiety may have proved significant. Similarly significant effects of alcohol consumption on reaction time performance may have been seen had participants achieved a BAC of 0.05

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PENDIX A State Anxiety form From the State Trait Anxiety Inventory eilberger et al., 1970)

SELF-EVALUATION QUESTIONNAIRE

Developed by C. D. Spielberger, R. L. Gorsuch and R. Lushene

STAI FORM X-1

NAME ____________________________________ DATE ________

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you *feel* right now, that is, *at this moment*. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1. I feel calm	1	2	3	4
2. I feel secure	1	2	3	4
3. I am tense	1	2	3	4
4. I am regretful	1	2	3	4
5. I feel at ease	1	2	3	4
6. I feel upset	1	2	3	4
7. I am presently worrying over possible misfortunes	1	2	3	4
8. I feel rested	1	2	3	4
9. I feel anxious	1	2	3	4
10. I feel comfortable	1	2	3	4
11. I feel self-confident	1	2	3	4
12. I feel nervous	1	2	3	4
13. I am jittery	1	2	3	4
14. I feel "high strung"	1	2	3	4
15. I am relaxed	1	2	3	4
16. I feel content	1	2	3	4
17. I am worried	1	2	3	4
18. I feel over-excited and "rattled"	1	2	3	4
19. I feel joyful	1	2	3	4
20. I feel pleasant	1	2	3	4

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APPENDIX B Medical History and Drinking Questionairre

University of Tasmania
Department of Psychology

Medical History Questionnaire

NAME..

AGE...PHONE.....................................

Do you; A. Smoke Cigarettes................................ Yes ☐ No ☐
B. Use or have experimented with either drugs or marijuana .... Yes ☐ No ☐

Have you ever been a patient in a Mental hospital?......... Yes ☐ No ☐

Have you ever been a patient in any other hospital ?......... Yes ☐ No ☐

HAVE YOU EVER HAD OR ARE YOU NOW SUFFERING FROM ANY OF THE FOLLOWING:

Fits or Convulsions.......................... Yes ☐ No ☐

Epilepsy....................................... Yes ☐ No ☐

Giddiness..................................... Yes ☐ No ☐

Concussion.................................... Yes ☐ No ☐

Severe Head Injury.......................... Yes ☐ No ☐

Loss of Consciousness....................... Yes ☐ No ☐

CURRENT MEDICATION

Are you taking any medications at present ? ...... Yes ☐ No ☐
If YES, which Drugs are you taking?
....
....

HEARING

Have you any hearing difficulties? ...... Yes ☐ No ☐
If YES, indicate hearing defects ....

DRINKING HISTORY

On how many days last week did you drink alcohol ?... None ☐
One or Two days ☐
Five or Six Days ☐
Every Day ☐

Do you usually drink... Never ☐
During the Week ☐
Friday Night ☐
Week Ends Only ☐

When you drink is it Normally................................ Light Beer ☐
Beer or Cider ☐
Wine ☐
Mixed spirits ☐
Straight Spirits ☐

On a day when you drink, how many drinks would you usually have?
One or Two ☐
Three to Five ☐
Five to Eight ☐
Eight to Twelve ☐
More than Twelve ☐

How long have you been drinking at this level ?............ Weeks ☐
Months ☐
Years ☐

Do you get drunk?... Never ☐
Rarely ☐
Once a Month ☐

Does your father get drunk?.................................... Never ☐
Rarely ☐
Once a Month ☐
Once a Week ☐
More Frequently ☐

Does your Mother get drunk?................................... Never ☐
Rarely ☐
Once a Month ☐
Once a Week ☐
More Frequently ☐

Do you have any relatives whom you would consider to be alcoholic?
Yes ☐ No ☐

If YES, How many and what relationship are they to you? ....
....
....

OTHER INFORMATION

How often do you smoke Cigarettes ?................. Never ☐
Less than 10 per day ☐
10 to 20 per day ☐
20 to 40 per day ☐
Over 40 per day ☐

Note:

It is a formal requirement of the Ethics Committee of the University of Tasmania that the information provided on this questionnaire be held under security to comply with confidentiality regulations and to protect your privacy. You can be assured that information will be available only to the principal researcher and not to any other party. The questionnaire will be destroyed following the completion of the project.

Thankyou for your participation.

APPENDIX C Raw data and analyses.

dI's Masters thesis

STAT. GENERAL MANOVA	Summary of all Effects; design: (dimast.sta) 1-SEX, 2-PRE/POST, 3-ALCY/N, 4-TOLDY/N					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
12	1	110.5563	18	63.4590	1.742167	.203405
13	1	20.3062	18	84.6257	.239954	.630156
23	1	174.3062	18	88.1201	1.978052	.176626
14	1	5.2562	18	55.5201	.094673	.761850
24	1	51.7562	18	16.3868	3.158410	.092438
34	1	31.5063	18	14.9924	2.101487	.164356
123	1	1.4063	18	88.1201	.015958	.900874
124	1	12.6563	18	16.3868	.772344	.391074
134	1	41.0062	18	14.9924	2.735143	.115496
234	1	2.2562	18	7.3868	.305443	.587288
1234	1	.1563	18	7.3868	.021153	.885980

STAT. GENERAL MANOVA	INTERACTION: 1 x 2 x 3 x 4 (dimast.sta) 1-SEX, 2-PRE/POST, 3-ALCY/N, 4-TOLDY/N				
Univar. Test	Sum of Squares	df	Mean Square	F	p-level
Effect	.1563	1	.156250	.021153	.885980
Error	132.9625	18	7.386806		

STAT. GENERAL MANOVA				Means (dimast.sta) F(1,18)=.02; p<.8860
SEX	PRE/POST	ALCY/N	TOLDY/N	Depend. Var.1
1	1	1	1	33.40000
1	1	1	2	33.80000
1	1	2	1	30.80000
1	1	2	2	31.10000
1	2	1	1	35.70000
1	2	1	2	34.60000
1	2	2	1	37.30000
1	2	2	2	36.80000
2	1	1	1	29.50000
2	1	1	2	33.90000
2	1	2	1	28.00000
2	1	2	2	28.00000
2	2	1	1	30.10000
2	2	1	2	30.50000
2	2	2	1	31.80000
2	2	2	2	29.00000

STAT. GENERAL MANOVA	MAIN EFFECT: SEX (dimast.sta) 1-SEX, 2-PRE/POST, 3-ALCY/N, 4-TOLDY/N				
Univar. Test	Sum of Squares	df	Mean Square	F	p-level
Effect	668.306	1	668.3063	3.282929	.086722
Error	3664.262	18	203.5701		

dI's Masters thesis

STAT. GENERAL MANOVA				Means (dimast.sta) F(1,18)=3.28; p<.0867
SEX	PRE/POST	ALCY/N	TOLDY/N	Depend. Var.1
1	....	....	....	34.18750
2	....	....	....	30.10000

STAT. GENERAL MANOVA	INTERACTION: 2 x 4 (dimast.sta) 1-SEX, 2-PRE/POST, 3-ALCY/N, 4-TOLDY/N				
Univar. Test	Sum of Squares	df	Mean Square	F	p-level
Effect	51.7562	1	51.75625	3.158410	.092438
Error	294.9625	18	16.38681		

STAT. GENERAL MANOVA				Means (unweighted) (dimast.sta) F(1,18)=3.16; p<.0924
SEX	PRE/POST	ALCY/N	TOLDY/N	Depend. Var.1
....	1	....	1	30.42500
....	1	....	2	31.70000
....	2	....	1	33.72500
....	2	....	2	32.72500

STAT. GENERAL MANOVA					Newman-Keuls test; Var.1 (dimast.sta) Probabilities for Post Hoc Tests INTERACTION: 2 x 4		
SEX	PRE/POST	ALCY/N	TOLDY/N		{1} 30.42500	{2} 31.70000	{3} 33.72500
....	1	....	1	{1}		.176147	.009220*
....	1	....	2	{2}	.176147		.091831
....	2	....	1	{3}	.009220*	.091831	
....	2	....	2	{4}	.051247	.272471	.283946

STAT. GENERAL MANOVA					Newman-Keuls test; Var.1 (dimast.sta) Probabilities for Post Hoc Tests INTERACTION: 2 x 4
SEX	PRE/POST	ALCY/N	TOLDY/N		{4} 32.72500
....	1	....	1	{1}	.051247
....	1	....	2	{2}	.272471
....	2	....	1	{3}	.283946
....	2	....	2	{4}	

data file: DIMAST.STA [30 cases with 109 variables]
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STAT. GENERAL MANOVA	Means (dimast.sta) 8 Variables					
SEX	FATNGNSA	FBTNGN	FATNGASA	FBTNGA	FATAGNSA	FBTAGN
G_1:1	33.40000	33.80000	30.80000	31.10000	35.70000	34.60000
G_2:2	29.50000	33.90000	28.00000	28.00000	30.10000	30.50000
All Groups	31.45000	33.85000	29.40000	29.55000	32.90000	32.55000

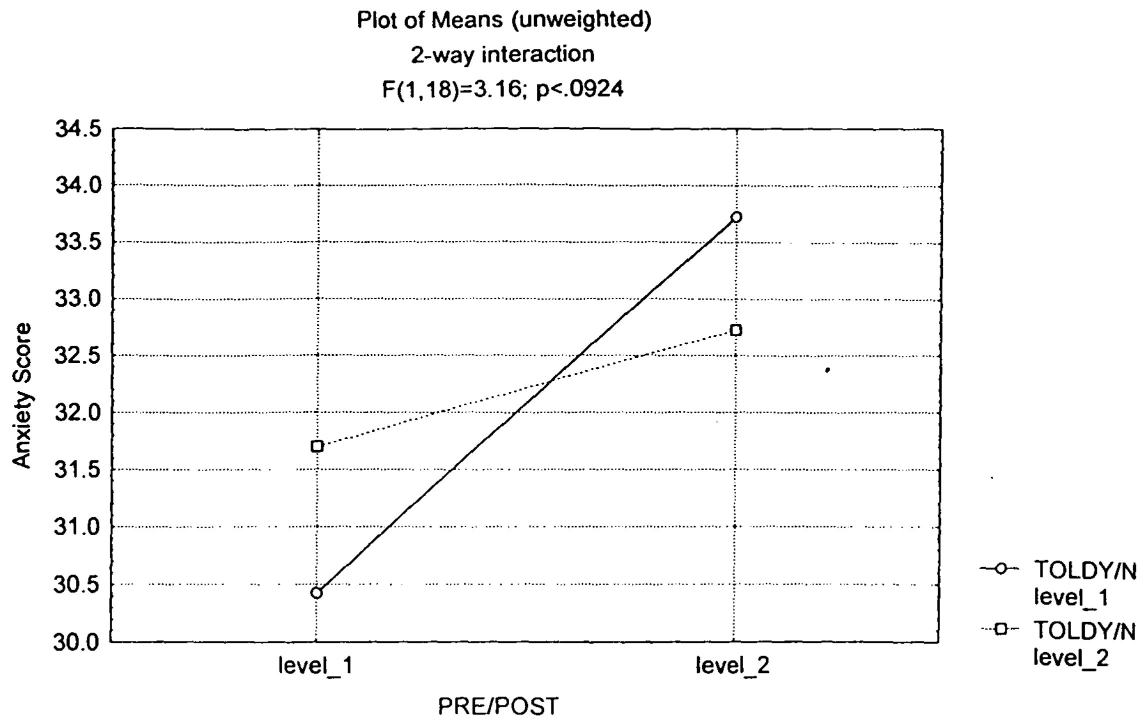
STAT. GENERAL MANOVA	Means (dimast.sta) 8 Variables		
SEX	FATAGASA	FBTAGA	Valid N
G_1:1	37.30000	36.80000	10
G_2:2	31.80000	29.00000	10
All Groups	34.55000	32.90000	20

STAT. GENERAL MANOVA	Standard Deviations (dimast.sta) 8 Variables					
SEX	FATNGNSA	FBTNGN	FATNGASA	FBTNGA	FATAGNSA	FBTAGN
G_1:1	7.275530	5.51362	7.420692	6.349978	13.68738	8.435375
G_2:2	5.720334	10.30049	4.988076	6.749485	9.68905	7.487026
All Groups	6.676550	8.04118	6.319560	6.573271	11.89383	8.042486

STAT. GENERAL MANOVA	Standard Deviations (dimast.sta) 8 Variables		
SEX	FATAGASA	FBTAGA	Valid N
G_1:1	8.246885	11.14351	10
G_2:2	6.545567	6.05530	10
All Groups	7.776314	9.60208	20

DESIGN: 4 - way ANOVA , fixed effects
DEPENDENT: 1 variable (Repeated Measure)
BETWEEN: 1-SEX (2): 1 2
WITHIN: 2-PRE/POST(2) x 3-ALCY/N(2) x 4-TOLDY/N(2)

STAT. GENERAL MANOVA	Summary of all Effects; design: (dimast.sta) 1-SEX, 2-PRE/POST, 3-ALCY/N, 4-TOLDY/N					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	668.3063	18	203.5701	3.282929	.086722
2	1	187.0562	18	63.4590	2.947670	.103159
3	1	47.3063	18	84.6257	.559006	.464318
4	1	.7563	18	55.5201	.013621	.908382


Plot of Means (unweighted)
2-way interaction
F(1,18)=3.16; p<.0924
Anxiety Score
34.5
34.0
33.5
33.0
32.5
32.0
31.5
31.0
30.5
30.0
level_1
level_2
PRE/POST
TOLDY/N level_1
TOLDY/N level_2

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VARIABLE SPECIFICATIONS:

No	Name	Format	MD Code	Long Label
24	FATAGNRH	8.3	-9999	
34	FATAGARH	8.3	-9999	
46	MATNGNRH	8.3	-9999	
56	MATNGARH	8.3	-9999	
2	SEX	8.3	-9999	1=female, 2=male

INDEPENDENT VARIABLES (between-groups factors):

SEX Number of Levels: 2 Codes: level 1: 1
level 2: 2

data file: DIMAST.STA [30 cases with 109 variables]
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VARIABLE SPECIFICATIONS:

No	Name	Format	MD Code	Long Label
4	FATNGNRH	8.3	-9999	
14	FATNGARH	8.3	-9999	
24	FATAGNRH	8.3	-9999	
34	FATAGARH	8.3	-9999	
2	SEX	8.3	-9999	1=female, 2=male

INDEPENDENT VARIABLES (between-groups factors):

SEX Number of Levels: 2 Codes: level 1: 1
level 2: 2

```
    DESIGN: 1 - way MANOVA        , fixed effects
 DEPENDENT: 4 variables:   FATNGNRH FATNGARH FATAGNRH FATAGARH
   BETWEEN: 1-SEX      ( 2):     1     2
    WITHIN: none
```

data file: DIMAST.STA [30 cases with 109 variables]
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VARIABLE SPECIFICATIONS:

No	Name	Format	MD Code	Long Label
4	FATNGNRH	8.3	-9999	
14	FATNGARH	8.3	-9999	
24	FATAGNRH	8.3	-9999	
34	FATAGARH	8.3	-9999	
2	SEX	8.3	-9999	1=female, 2=male

INDEPENDENT VARIABLES (between-groups factors):

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```
SEX          Number of Levels:    2     Codes:  level   1:    1
                                                level   2:    2

     DESIGN: 3 - way ANOVA         , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN: 1-SEX      ( 2):     1     2
     WITHIN: 2-ALCY/N(2) x 3-TOLDY/N(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (dimast.sta) 1-SEX, 2-ALCY/N, 3-TOLDY/N					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	495.013	18	15902.70	.031128	.861926
2	1	812.813	18	1253.48	.648445	.431178
3	1	127.512	18	1888.60	.067517	.797936
12	1	195.313	18	1253.48	.155816	.697676
13	1	1720.512	18	1888.60	.911001	.352492
23	1	475.313	18	804.52	.590804	.452076
123	1	.112	18	804.52	.000140	.990695

STAT. GENERAL MANOVA			Means (dimast.sta) F(1,18)=.00; p<.9907
SEX	ALCY/N	TOLDY/N	Depend. Var.1
1	1	1	629.9000
1	1	2	631.7000
1	2	1	621.7000
1	2	2	633.4000
2	1	1	647.2000
2	1	2	630.6000
2	2	1	632.9000
2	2	2	625.9000

data file: DIMAST.STA [30 cases with 109 variables]
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VARIABLE SPECIFICATIONS:

No	Name	Format	MD Code	Long Label
5	FATNGNRF	8.3	-9999	
15	FATNGARF	8.3	-9999	
25	FATAGNRF	8.3	-9999	
35	FATAGARF	8.3	-9999	
2	SEX	8.3	-9999	1=female, 2=male

INDEPENDENT VARIABLES (between-groups factors):

```
SEX          Number of Levels:    2     Codes:  level   1:    1
                                                level   2:    2

     DESIGN: 3 - way ANOVA         , fixed effects
```

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DEPENDENT: 1 variable (Repeated Measure)
BETWEEN: 1-SEX (2): 1 2
WITHIN: 2-ALCY/N(2) x 3-TOLDY/N(2)

STAT. GENERAL MANOVA	Summary of all Effects; design: (dimast.sta) 1-SEX, 2-ALCY/N, 3-TOLDY/N					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	5271.93	17	24629.59	.214049	.649479
2	1	7380.27	17	4252.86	1.735364	.205207
3	1	1074.49	17	5793.74	.185457	.672135
12	1	678.95	17	4252.86	.159646	.694458
13	1	13730.33	17	5793.74	2.369856	.142104
23	1	1414.96	17	2525.75	.560214	.464404
123	1	9331.01	17	2525.75	3.694355	.071520

STAT. GENERAL MANOVA	INTERACTION: 1 x 2 x 3 (dimast.sta) 1-SEX, 2-ALCY/N, 3-TOLDY/N				
Univar. Test	Sum of Squares	df	Mean Square	F	p-level
Effect	9331.01	1	9331.012	3.694355	.071520
Error	42937.73	17	2525.749		

STAT. GENERAL MANOVA			Means (dimast.sta) F(1,17)=3.69; p<.0715
SEX	ALCY/N	TOLDY/N	Depend. Var.1
1	1	1	647.7778
1	1	2	636.3333
1	2	1	591.2222
1	2	2	641.4445
2	1	1	663.2000
2	1	2	642.3000
2	2	1	663.0000
2	2	2	615.0000

STAT. GENERAL MANOVA				Newman-Keuls test; Var.1 (dimast.sta) Probabilities for Post Hoc Tests INTERACTION: 1 x 2 x 3			
SEX	ALCY/N	TOLDY/N		{1} 647.7778	{2} 636.3333	{3} 591.2222	{4} 641.4445
1	1	1	{1}		.959045	.194369	.959570
1	1	2	{2}	.959045		.154478	.827586
1	2	1	{3}	.194369	.154478		.170257
1	2	2	{4}	.959570	.827586	.170257	
2	1	1	{5}	.785009	.847714	.090927	.876489
2	1	2	{6}	.815443	.964033	.222266	.971007
2	2	1	{7}	.518742	.775861	.075797	.787602
2	2	2	{8}	.624287	.368672	.317708	.500627

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STAT. GENERAL MANOVA				Newman-Keuls test; Var.1 (dimast.sta) Probabilities for Post Hoc Tests INTERACTION: 1 x 2 x 3			
SEX	ALCY/N	TOLDY/N		{5} 663.2000	{6} 642.3000	{7} 663.0000	{8} 615.0000
1	1	1	{1}	.785009	.815443	.518742	.624287
1	1	2	{2}	.847714	.964033	.775861	.368672
1	2	1	{3}	.090927	.222266	.075797	.317708
1	2	2	{4}	.876489	.971007	.787602	.500627
2	1	1	{5}		.802417	.993296	.401766
2	1	2	{6}	.802417		.649844	.645777
2	2	1	{7}	.993296	.649844		.342708
2	2	2	{8}	.401766	.645777	.342708	

data file: DIMAST.STA [30 cases with 109 variables]
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VARIABLE SPECIFICATIONS:

No	Name	Format	MD Code	Long Label
6	FATNGNH	8.3	-9999	
16	FATNGAH	8.3	-9999	
26	FATAGNH	8.3	-9999	
36	FATAGAH	8.3	-9999	
2	SEX	8.3	-9999	1=female, 2=male

INDEPENDENT VARIABLES (between-groups factors):

SEX Number of Levels: 2 Codes: level 1: 1
level 2: 2

DESIGN: 3 - way ANOVA , fixed effects
DEPENDENT: 1 variable (Repeated Measure)
BETWEEN: 1-SEX (2): 1 2
WITHIN: 2-ALCY/N(2) x 3-TOLDY/N(2)

data file: DIMAST.STA [30 cases with 109 variables]
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VARIABLE SPECIFICATIONS:

No	Name	Format	MD Code	Long Label
7	FATNGNM	8.3	-9999	
17	FATNGAM	10.3	-9999	
27	FATAGNM	8.3	-9999	
37	FATAGAM	8.3	-9999	
2	SEX	8.3	-9999	1=female, 2=male

INDEPENDENT VARIABLES (between-groups factors):

SEX Number of Levels: 2 Codes: level 1: 1

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level 2: 2

DESIGN: 3 - way ANOVA , fixed effects
DEPENDENT: 1 variable (Repeated Measure)
BETWEEN: 1-SEX (2): 1 2
WITHIN: 2-ALCY/N(2) x 3-TOLDY/N(2)

STAT. GENERAL MANOVA	Summary of all Effects; design: (dimast.sta) 1-SEX, 2-ALCY/N, 3-TOLDY/N					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	117.6125	18	60.00139	1.960163	.178496
2	1	23.1125	18	42.99028	.537622	.472862
3	1	99.0125	18	45.15139	2.192900	.155940
12	1	70.3125	18	42.99028	1.635544	.217181
13	1	127.5125	18	45.15139	2.824110	.110129
23	1	86.1125	18	41.36250	2.081898	.166232
123	1	94.6125	18	41.36250	2.287398	.147787

STAT. GENERAL MANOVA	INTERACTION: 1 x 2 x 3 (dimast.sta) 1-SEX, 2-ALCY/N, 3-TOLDY/N				
Univar. Test	Sum of Squares	df	Mean Square	F	p-level
Effect	94.6125	1	94.61250	2.287398	.147787
Error	744.5250	18	41.36250		

STAT. GENERAL MANOVA			Means (dimast.sta) F(1,18)=2.29; p<.1478
SEX	ALCY/N	TOLDY/N	Depend. Var.1
1	1	1	2.90000
1	1	2	3.40000
1	2	1	1.60000
1	2	2	10.60000
2	1	1	2.70000
2	1	2	2.50000
2	2	1	2.00000
2	2	2	1.60000

data file: DIMAST.STA [30 cases with 109 variables]
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VARIABLE SPECIFICATIONS:

No	Name	Format	MD Code	Long Label
8	FATNGNFA	8.3	-9999	
18	FATNGAFA	8.3	-9999	
28	FATAGNFA	8.3	-9999	
38	FATAGAFA	8.3	-9999	

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No	Name	Format	MD Code	Long Label
2	SEX	8.3	-9999	1=female, 2=male

INDEPENDENT VARIABLES (between-groups factors):

SEX Number of Levels: 2 Codes: level 1: 1
level 2: 2

DESIGN: 3 - way ANOVA , fixed effects
DEPENDENT: 1 variable (Repeated Measure)
BETWEEN: 1-SEX (2): 1 2
WITHIN: 2-ALCY/N(2) x 3-TOLDY/N(2)

STAT. GENERAL MANOVA	Summary of all Effects; design: (dimast.sta) 1-SEX, 2-ALCY/N, 3-TOLDY/N					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	84.0500	18	310.8445	.270392	.609405
2	1	72.2000	18	39.9722	1.806254	.195655
3	1	39.2000	18	46.6833	.839700	.371597
12	1	57.8000	18	39.9722	1.446004	.244751
13	1	45.0000	18	46.6833	.963941	.339209
23	1	54.4500	18	42.6556	1.276504	.273382
123	1	101.2500	18	42.6556	2.373665	.140793

STAT. GENERAL MANOVA			Means (dimast.sta) F(1,18)=2.37; p<.1408
SEX	ALCY/N	TOLDY/N	Depend. Var.1
1	1	1	4.30000
1	1	2	3.30000
1	2	1	4.00000
1	2	2	10.80000
2	1	1	7.30000
2	1	2	7.80000
2	2	1	8.10000
2	2	2	7.40000

data file: DIMAST.STA [30 cases with 109 variables]
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VARIABLE SPECIFICATIONS:

No	Name	Format	MD Code	Long Label
9	FATNGNCR	8.3	-9999	
19	FATNGACR	8.3	-9999	
29	FATAGNCR	8.3	-9999	
39	FATAGACR	8.3	-9999	
2	SEX	8.3	-9999	1=female, 2=male

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INDEPENDENT VARIABLES (between-groups factors):

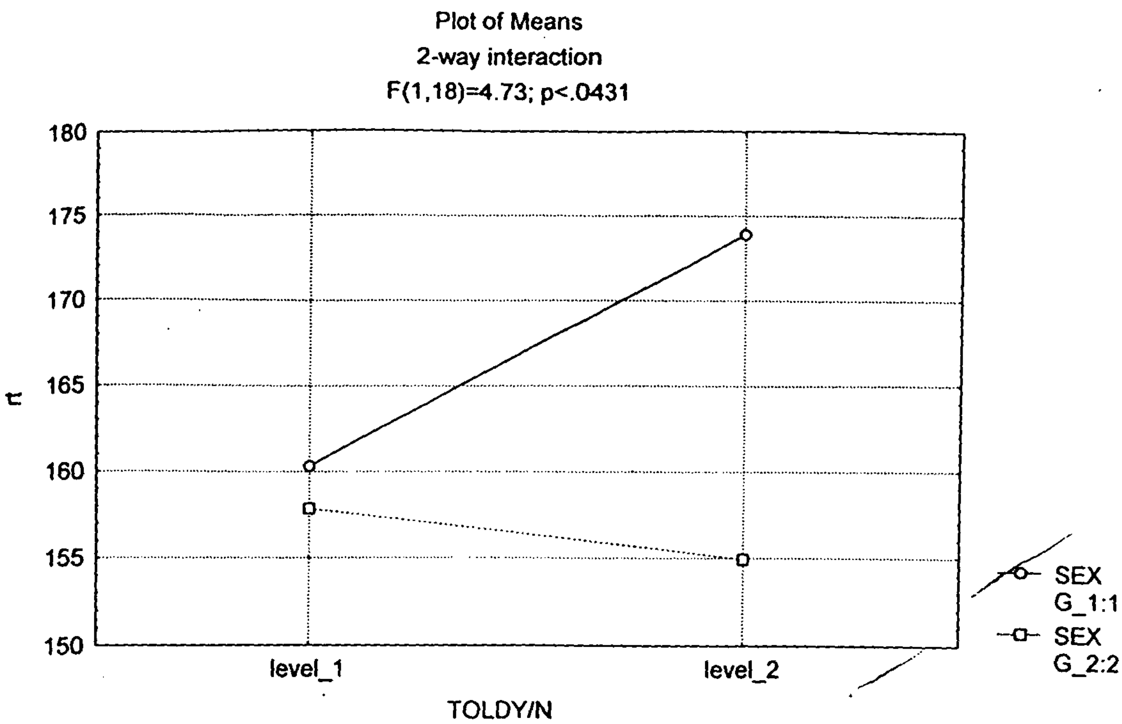
SEX Number of Levels: 2 Codes: level 1: 1
level 2: 2

DESIGN: 3 - way ANOVA , fixed effects
DEPENDENT: 1 variable (Repeated Measure)
BETWEEN: 1-SEX (2): 1 2
WITHIN: 2-ALCY/N(2) x 3-TOLDY/N(2)

STAT. GENERAL MANOVA	Summary of all Effects; design: (dimast.sta) 1-SEX, 2-ALCY/N, 3-TOLDY/N					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	2289.800	18	864.9833	2.647219	.121109
2	1	288.800	18	526.2722	.548765	.468377
3	1	572.450	18	287.5444	1.990823	.175305
12	1	1216.800	18	526.2722	2.312111	.145741
13	1*	1361.250*	18*	287.5444*	4.734051*	.043137*
23	1	1201.250	18	664.3444	1.808173	.195428
123	1	832.050	18	664.3444	1.252438	.277800

STAT. GENERAL MANOVA	INTERACTION: 1 x 2 x 3 (dimast.sta) 1-SEX, 2-ALCY/N, 3-TOLDY/N				
Univar. Test	Sum of Squares	df	Mean Square	F	p-level
Effect	832.05	1	832.0500	1.252438	.277800
Error	11958.20	18	664.3444		

STAT. GENERAL MANOVA			Means (dimast.sta) F(1,18)=1.25; p<.2778
SEX	ALCY/N	TOLDY/N	Depend. Var.1
1	1	1	161.6000
1	1	2	161.0000
1	2	1	159.0000
1	2	2	186.8000
2	1	1	160.5000
2	1	2	156.3000
2	2	1	155.2000
2	2	2	153.6000


Plot of Means
2-way interaction
F(1,18)=4.73; p<.0431
180
175
170
165
160
155
150
rt
level_1
level_2
TOLDY/N
SEX G_1:1
SEX G_2:2

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STAT. GENERAL MANOVA	INTERACTION: 1 x 3 (dimast.sta) 1-SEX, 2-ALCY/N, 3-TOLDY/N				
Univar. Test	Sum of Squares	df	Mean Square	F	p-level
Effect	1361.250	1	1361.250	4.734051	.043137
Error	5175.800	18	287.544		

STAT. GENERAL MANOVA			Means (dimast.sta) F(1,18)=4.73; p<.0431
SEX	ALCY/N	TOLDY/N	Depend. Var.1
1	....	1	160.3000
1	....	2	173.9000
2	....	1	157.8500
2	....	2	154.9500

STAT. GENERAL MANOVA					Newman-Keuls test; Var.1 (dimast.sta) Probabilities for Post Hoc Tests INTERACTION: 1 x 3			
	SEX	ALCY/N	TOLDY/N		{1} 160.3000	{2} 173.9000	{3} 157.8500	{4} 154.9500
F	1	....	1	{1}		.020836*	.653353	.587739
	1	....	2	{2}	.020836*		.020383*	.011668*
M	2	....	1	{3}	.653353	.020383*		.595396
	2	....	2	{4}	.587739	.011668*	.595396	

data file: DIMAST.STA [30 cases with 109 variables]
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VARIABLE SPECIFICATIONS:

No	Name	Format	MD Code	Long Label
10	FATNGNSI	8.3	-9999	
20	FATNGASI	8.3	-9999	
30	FATAGNSI	8.3	-9999	
40	FATAGASI	8.3	-9999	
2	SEX	8.3	-9999	1=female, 2=male

INDEPENDENT VARIABLES (between-groups factors):

SEX Number of Levels: 2 Codes: level 1: 1
level 2: 2

DESIGN: 3 - way ANOVA , fixed effects
DEPENDENT: 1 variable (Repeated Measure)
BETWEEN: 1-SEX (2): 1 2
WITHIN: 2-ALCY/N(2) x 3-TOLDY/N(2)

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data file: DIMAST.STA [30 cases with 109 variables]
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	1 SS_INIT	2 SEX	3 FATNGNSA	4 FATNGNRH	5 FATNGNRF	6 FATNGNH	7 FATNGNM	8 FATNGNFA	9 FATNGNCR
1	MP	1.000	39.000	632.000	700.000	40.000	3.000	7.000	159.000
2	DD	1.000	31.000	675.000	709.000	40.000	6.000	3.000	156.000
3	SH	1.000	25.000	509.000	469.000	40.000	1.000	2.000	150.000
4	SP	1.000	24.000	738.000	780.000	40.000	1.000	4.000	156.000
5	NT	1.000	35.000	707.000	576.000	40.000	7.000	2.000	185.000
6	GP	1.000	31.000	626.000	554.000	40.000	2.000	1.000	168.000
7	MZ	1.000	40.000	677.000	773.000	40.000	4.000	4.000	167.000
8	EH	1.000	47.000	630.000	571.000	40.000	1.000	2.000	158.000
9	SLA	1.000	35.000	545.000	629.000	40.000	2.000	15.000	147.000
10	DP	1.000	27.000	560.000	645.000	40.000	2.000	3.000	170.000
11	#MC	2.000	41.000	592.000	603.000	40.000	0.000	4.000	155.000
12	#EFG	2.000	33.000	685.000	831.000	40.000	0.000	8.000	151.000
13	#TO	2.000	29.000	557.000	578.000	40.000	1.000	30.000	150.000
14	#WO	2.000	33.000	642.000	529.000	40.000	7.000	2.000	180.000
15	#DD	2.000	24.000	666.000	730.000	40.000	4.000	7.000	164.000
16	#MJ	2.000	26.000	688.000	575.000	40.000	2.000	13.000	149.000
17	*NT	2.000	20.000	752.000	846.000	40.000	10.000	1.000	192.000
18	#DW	2.000	30.000	627.000	739.000	40.000	1.000	2.000	150.000
19	#PO	2.000	28.000	613.000	547.000	40.000	1.000	3.000	157.000
20	#NP	2.000	31.000	650.000	654.000	40.000	1.000	3.000	157.000
21									
22									
23									
24									
25									
26									
27									
28									
29									
30									

	10 FATNGNSI	11 FATNGNBA	12 FBTNGN	13 FATNGASA	14 FATNGARH	15 FATNGARF	16 FATNGAH	17 FATNGAM
1	0.000	0.000	33.000	31.000	505.000	619.000	40.000	1.000
2	0.000	0.000	35.000	37.000	636.000	601.000	40.000	3.000
3	0.000	0.000	26.000	25.000	534.000	581.000	40.000	1.000
4	0.000	0.000	29.000	24.000	764.000	822.000	40.000	8.000
5	0.000	1.000	38.000	36.000	679.000		40.000	10.000
6	0.000	0.000	32.000	25.000	677.000	650.000	40.000	2.000
7	0.000	0.000	41.000	44.000	630.000	635.000	40.000	1.000
8	0.000	0.000	42.000	37.000	630.000	631.000	40.000	4.000
9	0.000	0.000	35.000	21.000	562.000	586.000	40.000	1.000
10	0.000	0.000	27.000	28.000	700.000	602.000	40.000	3.000
11	0.000	0.000	45.000	27.000	549.000	495.000	40.000	1.000
12	0.000	0.000	31.000	25.000	690.000	789.000	40.000	2.000
13	0.000	0.000	41.000	30.000	526.000	593.000	40.000	1.000
14	0.000	0.000	31.000	23.000	649.000	562.000	40.000	2.000
15	0.000	0.000	25.000	24.000	695.000	838.000	40.000	3.000
16	0.000	0.000	42.000	34.000	632.000	692.000	40.000	3.000
17	0.000	0.000	20.000	20.000	763.000	716.000	40.000	9.000
18	0.000	0.000	30.000	35.000	643.000	656.000	40.000	2.000
19	0.000	0.000	23.000	32.000	631.000	576.000	40.000	1.000
20	1.000	1.000	51.000	30.000	528.000	506.000	40.000	1.000
21								
22								
23								
24								
25								

	10 FATNGNSI	11 FATNGNBA	12 FBTNGN	13 FATNGASA	14 FATNGARH	15 FATNGARF	16 FATNGAH	17 FATNGAM
26								
27								
28								
29								
30								

	18 FATNGAFA	19 FATNGACR	20 FATNGASI	21 FATNGABA	22 FBTNGA	23 FATAGNSA	24 FATAGNRH	25 FATAGNRF	26 FATAGNH
1	8.000	152.000	3.000	3.000	28.000	33.000	548.000	601.000	40.000
2	1.000	165.000	1.000	1.000	32.000	32.000	685.000	633.000	40.000
3	3.000	157.000	1.000	1.000	34.000	25.000	509.000	466.000	40.000
4	3.000	181.000	2.000	1.000	27.000	23.000	761.000	664.000	40.000
5	0.000	192.000	1.000	1.000	33.000	37.000	659.000		40.000
6	2.000	114.000	0.000	0.000	30.000	32.000	593.000	550.000	40.000
7	4.000	156.000	2.000	1.000	46.000	39.000	629.000	644.000	40.000
8	4.000	167.000	1.000	0.000	27.000	72.000	641.000	634.000	40.000
9	4.000	156.000	0.000	0.000	22.000	29.000	542.000	564.000	40.000
10	4.000	170.000	1.000	1.000	32.000	35.000	650.000	565.000	40.000
11	3.000	157.000	0.000	2.000	25.000	23.000	625.000	713.000	40.000
12	4.000	150.000	0.000	0.000	28.000	23.000	642.000	801.000	40.000
13	46.000	114.000	0.000	0.000	39.000	32.000	594.000	536.000	40.000
14	1.000	161.000	0.000	0.000	26.000	28.000	699.000	614.000	40.000
15	5.000	161.000	1.000	1.000	21.000	25.000	674.000	835.000	40.000
16	6.000	160.000	2.000	2.000	39.000	23.000	654.000	637.000	40.000
17	5.000	184.000	0.000	0.000	20.000	42.000	721.000	715.000	40.000
18	4.000	160.000	1.000	1.000	32.000	52.000	580.000	734.000	40.000
19	2.000	158.000	0.000	0.000	23.000	24.000	559.000	477.000	40.000
20	2.000	158.000	0.000	0.000	27.000	29.000	581.000	568.000	40.000
21									
22									
23									
24									
25									
26									
27									
28									
29									
30									

	27 FATAGNM	28 FATAGNFA	29 FATAGNCR	30 FATAGNSI	31 FATAGNBA	32 FBTAGN	33 FATAGASA	34 FATAGARH	35 FATAGARF
1	0.000	15.000	144.000	0.000	1.000	32.000	35.000	501.000	645.000
2	2.000	5.000	164.000	2.000	2.000	25.000	32.000	672.000	648.000
3	1.000	3.000	157.000	1.000	1.000	28.000	24.000	513.000	545.000
4	3.000	2.000	170.000	0.000	2.000	24.000	42.000	747.000	762.000
5	3.000	0.000	166.000	3.000	2.000	45.000	36.000	636.000	785.000
6	2.000	2.000	160.000	1.000	2.000	29.000	33.000	697.000	558.000
7	1.000	4.000	156.000	1.000	1.000	42.000	51.000	623.000	596.000
8	1.000	2.000	158.000	1.000	1.000	48.000	50.000	709.000	737.000
9	1.000	5.000	155.000	0.000	1.000	35.000	35.000	566.000	582.000
10	2.000	2.000	160.000	2.000	2.000	38.000	35.000	670.000	700.000
11	2.000	2.000	160.000	0.000	1.000	22.000	40.000	595.000	552.000
12	0.000	8.000	151.000	2.000	2.000	23.000	28.000	590.000	676.000
13	5.000	43.000	133.000	3.000	3.000	33.000	42.000	536.000	530.000
14	2.000	1.000	161.000	1.000	1.000	27.000	35.000	690.000	690.000
15	2.000	9.000	153.000	1.000	1.000	25.000	27.000	677.000	625.000
16	3.000	5.000	161.000	0.000	0.000	27.000	28.000	663.000	758.000
17	3.000	2.000	164.000	0.000	0.000	43.000	20.000	697.000	600.000
18	1.000	5.000	155.000	1.000	0.000	40.000	34.000	663.000	660.000
19	1.000	3.000	157.000	1.000	1.000	27.000	30.000	579.000	534.000
20	1.000	3.000	157.000	1.000	1.000	38.000	34.000	569.000	525.000

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	27 FATAGNM	28 FATAGNFA	29 FATAGNCR	30 FATAGNSI	31 FATAGNBA	32 FBTAGN	33 FATAGASA	34 FATAGARH	35 FATAGARF
21									
22									
23									
24									
25									
26									
27									
28									
29									
30									

	36 FATAGAH	37 FATAGAM	38 FATAGAFA	39 FATAGACR	40 FATAGASI	41 FATAGABA	42 FBTAGA	43 NEWVAR	44 SS_IN
1	40.000	1.000	7.000	153.000	2.000	2.000	32.000	#	
2	40.000	1.000	1.000	159.000	0.000	0.000	26.000		
3	40.000	1.000	4.000	156.000	3.000	5.000	29.000		
4	40.000	4.000	3.000	168.000	1.000	2.000	58.000	#	
5	40.000	35.000	19.000	277.000	3.000	2.000	37.000	#	
6	40.000	54.000	54.000	310.000	2.000	2.000	28.000		
7	40.000	1.000	3.000	157.000	1.000	1.000	51.000		
8	40.000	6.000	3.000	174.000	2.000	4.000	27.000		
9	40.000	0.000	9.000	150.000	2.000	1.000	34.000		
10	40.000	3.000	5.000	164.000	3.000	4.000	46.000		
11	40.000	1.000	2.000	150.000	0.000	3.000	34.000		
12	40.000	1.000	6.000	154.000	2.000	2.000	29.000		
13	40.000	0.000	44.000	115.000	3.000	3.000	39.000		
14	40.000	3.000	2.000	164.000	1.000	1.000	33.000		
15	40.000	5.000	4.000	172.000	1.000	1.000	23.000		
16	40.000	1.000	6.000	154.000	2.000	2.000	29.000		
17	40.000	1.000	2.000	158.000	1.000	2.000	21.000		
18	40.000	1.000	4.000	159.000	3.000	2.000	31.000		
19	40.000	1.000	2.000	150.000	2.000	2.000	20.000		
20	40.000	2.000	2.000	160.000	1.000	1.000	31.000		
21									
22									
23									
24									
25									
26									
27									
28									
29									
30									

	45 MATNGNSA	46 MATNGNRH	47 MATNGNRF	48 MATNGNH	49 MATNGNM	50 MATNGNFA	51 MATNGNCR	52 MATNGNSI	53 MATNGNBA
1									
2									
3									
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AT. ERAL OVA	Summary of all Effects; design: (dimast2.sta) 1-SEX, 2-PRESENT, 3-TOLDN/Y, 4-ALCN/Y, 5-PRE/POST					
ect	df Effect	MS Effect	df Error	MS Error	F	p-level
	1	852.5018	12	243.0964	3.506847	.085675
	3	135.6577	12	243.0964	.558041	.652697
	1	52.5018	12	64.1276	.818709	.383365
	1	24.3107	12	91.8047	.264809	.616181
	1	12.0607	12	61.1797	.197135	.664944
	3	109.4435	12	243.0964	.450206	.721797
	1	108.5460	12	64.1276	1.692656	.217679
	3	93.9058	12	64.1276	1.464358	.273570
	1	.2224	12	91.8047	.002423	.961552
	3	23.3958	12	91.8047	.254844	.856424
	1	81.8401	12	103.3359	.791981	.390994
	1	.0165	12	61.1797	.000270	.987150
	3	74.5724	12	61.1797	1.218908	.345206
	1	4.7813	12	18.8568	.253556	.623698
	1	41.9136	12	16.6380	2.519146	.138457
3	3	12.7272	12	64.1276	.198467	.895422
4	3	74.1577	12	91.8047	.807777	.513520
4	1	3.3989	12	103.3359	.032892	.859112
4	3	90.7907	12	103.3359	.878597	.479444
5	3	8.2431	12	61.1797	.134735	.937437
5	1	2.7960	12	18.8568	.148273	.706929
5	3	11.3958	12	18.8568	.604336	.624636
5	1	7.2960	12	16.6380	.438511	.520357
5	3	2.8224	12	16.6380	.169637	.914827
5	1	.1489	12	6.6484	.022396	.883525
34	3	10.6835	12	103.3359	.103386	.956507
35	3	33.1022	12	18.8568	1.755453	.209047
45	3	38.9851	12	16.6380	2.343134	.124589
45	1	4.4136	12	6.6484	.663856	.431076
45	3	7.2133	12	6.6484	1.084961	.392625
345	3	8.6657	12	6.6484	1.303415	.318472

AT. NERAL NOVA	Summary of all Effects; design: (dimast2.sta) 1-SEX, 2-PRESENT, 3-TOLDN/Y, 4-ALCN/Y					
fect	df Effect	MS Effect	df Error	MS Error	F	p-level
	1	1219.765	12	19267.00	.063308	.805598
	3	8849.163	12	19267.00	.459291	.715788
	1	1690.015	12	1133.00	1.491628	.245416
	1	108.765	12	2647.56	.041081	.842777
	3	5270.552	12	19267.00	.273553	.843333
	1	1948.471	12	1133.00	1.719745	.214264
	3	549.242	12	1133.00	.484768	.699112
	1	2484.132	12	2647.56	.938271	.351841
	3	589.762	12	2647.56	.222757	.878741
	1	218.882	12	528.23	.414370	.531870
3	3	2003.607	12	1133.00	1.768409	.206598
4	3	316.762	12	2647.56	.119643	.946796
4	1	481.779	12	528.23	.912065	.358411
4	3*	2786.111*	12*	528.23*	5.274436*	.014973*
34	3	1131.357	12	528.23	2.141792	.148176

AT. NERAL NOVA	INTERACTION: 2 x 3 x 4 (dimast2.sta) 1-SEX, 2-PRESENT, 3-TOLDN/Y, 4-ALCN/Y				
ivar. st	Sum of Squares	df	Mean Square	F	p-level
fect	8358.333	3	2786.111	5.274436	.014973
ror	6338.750	12	528.229		

Di's Masters results

STAT. GENERAL MANOVA				Means (unweighted) (dimast2.sta) F(3,12)=5.27; p<.0150
SEX	PRESENT	TOLDN/Y	ALCN/Y	Depend. Var.1
....	1	1	1	653.7500
....	1	1	2	616.0000
....	1	2	1	617.2500
....	1	2	2	632.2500
....	2	1	1	647.5000
....	2	1	2	644.5000
....	2	2	1	627.7500
....	2	2	2	604.5000
....	3	1	1	591.2500
....	3	1	2	606.5000
....	3	2	1	601.0000
....	3	2	2	595.0000
....	4	1	1	643.8750
....	4	1	2	673.8750
....	4	2	1	663.2500
....	4	2	2	651.5000

STAT. GENERAL MANOVA					Newman-Keuls test; Var.1 (dimast2.sta) Probabilities for Post Hoc Tests INTERACTION: 2 x 3 x 4				
SEX	PRESENT	TOLDN/Y	ALCN/Y		{1} 653.7500	{2} 616.0000	{3} 617.2500	{4} 632.2500	{5} 647.5000
....	1	1	1	{1}		.374410	.367832	.753056	.919087
....	1	1	2	{2}	.374410		.938777	.740616	.471538
....	1	2	1	{3}	.367832	.938777		.624960	.445646
....	1	2	2	{4}	.753056	.740616	.624960		.774751
....	2	1	1	{5}	.919087	.471538	.445646	.774751	
....	2	1	2	{6}	.935852	.505000	.462288	.727793	.853700
....	2	2	1	{7}	.665934	.746075	.521891	.782249	.729056
....	2	2	2	{8}	.172142	.755113	.852558	.531254	.241216
....	3	1	1	{9}	.065222	.638983	.665934	.286920	.098519
....	3	1	2	{10}	.185307	.561679	.781771	.513927	.251967
....	3	2	1	{11}	.137503	.783055	.840905	.480108	.198461
....	3	2	2	{12}	.085465	.685006	.727580	.346388	.126876
....	4	1	1	{13}	.968930	.441477	.378009	.479166	.971930
....	4	1	2	{14}	.440323	.075301	.075169	.238373	.492152
....	4	2	1	{15}	.561679	.185307	.183725	.488597	.757850
....	4	2	2	{16}	.890020	.397706	.383685	.746234	.805876

STAT. GENERAL MANOVA					Newman-Keuls test; Var.1 (dimast2.sta) Probabilities for Post Hoc Tests INTERACTION: 2 x 3 x 4				
SEX	PRESENT	TOLDN/Y	ALCN/Y		{6} 644.5000	{7} 627.7500	{8} 604.5000	{9} 591.2500	{10} 606.5000
....	1	1	1	{1}	.935852	.665934	.172142	.065222	.185307
....	1	1	2	{2}	.505000	.746075	.755113	.638983	.561679
....	1	2	1	{3}	.462288	.521891	.852558	.665934	.781771
....	1	2	2	{4}	.727793	.782249	.531254	.286920	.513927
....	2	1	1	{5}	.853700	.729056	.241216	.098519	.251967
....	2	1	2	{6}		.723080	.274977	.118103	.281280
....	2	2	1	{7}	.723080		.603594	.367832	.559491
....	2	2	2	{8}	.274977	.603594		.838024	.902175
....	3	1	1	{9}	.118103	.367832	.838024		.868315
....	3	1	2	{10}	.281280	.559491	.902175	.868315	
....	3	2	1	{11}	.230743	.566847	.829699	.816046	.936675
....	3	2	2	{12}	.150268	.430417	.824345	.817768	.886187
....	4	1	1	{13}	.969457	.582803	.249268	.111373	.247558
....	4	1	2	{14}	.474994	.181607	.030198*	.011154*	.032997*
....	4	2	1	{15}	.763078	.397706	.077293	.028301*	.084099
....	4	2	2	{16}	.899722	.674889	.189606	.073910	.201437

STAT. GENERAL MANOVA					Newman-Keuls test; Var.1 (dimast2.sta) Probabilities for Post Hoc Tests INTERACTION: 2 x 3 x 4				
SEX	PRESENT	TOLDN/Y	ALCN/Y		{11} 601.0000	{12} 595.0000	{13} 643.8750	{14} 673.8750	{15} 663.2500
....	1	1	1	{1}	.137503	.085465	.968930	.440323	.561679
....	1	1	2	{2}	.783055	.685006	.441477	.075301	.185307
....	1	2	1	{3}	.840905	.727580	.378009	.075169	.183725
....	1	2	2	{4}	.480108	.346388	.479166	.238373	.488597
....	2	1	1	{5}	.198461	.126876	.971930	.492152	.757850
....	2	1	2	{6}	.230743	.150268	.969457	.474994	.763078
....	2	2	1	{7}	.566847	.430417	.582803	.181607	.397706
....	2	2	2	{8}	.829699	.824345	.249268	.030198*	.077293
....	3	1	1	{9}	.816046	.817768	.111373	.011154*	.028301*
....	3	1	2	{10}	.936675	.886187	.247558	.032997*	.084099
....	3	2	1	{11}		.712797	.212950	.023690*	.060824
....	3	2	2	{12}	.712797		.140096	.014577*	.037238*
....	4	1	1	{13}	.212950	.140096		.523126	.820546
....	4	1	2	{14}	.023690*	.014577*	.523126		.517036
....	4	2	1	{15}	.060824	.037238*	.820546	.517036	
....	4	2	2	{16}	.153283	.096172	.962304	.519119	.746075

STAT. GENERAL MANOVA					Newman-Keuls test; Var.1 (dimast2.sta) Probabilities for Post Hoc Tests INTERACTION: 2 x 3 x 4
SEX	PRESENT	TOLDN/Y	ALCN/Y		{16} 651.5000
....	1	1	1	{1}	.890020
....	1	1	2	{2}	.397706
....	1	2	1	{3}	.383685
....	1	2	2	{4}	.746234
....	2	1	1	{5}	.805876
....	2	1	2	{6}	.899722
....	2	2	1	{7}	.674889
....	2	2	2	{8}	.189606
....	3	1	1	{9}	.073910
....	3	1	2	{10}	.201437
....	3	2	1	{11}	.153283
....	3	2	2	{12}	.096172
....	4	1	1	{13}	.962304
....	4	1	2	{14}	.519119
....	4	2	1	{15}	.746075
....	4	2	2	{16}	

STAT. GENERAL MANOVA	Summary of all Effects; design: (dimast2.sta) 1-SEX, 2-PRESENT					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	627.200	72	4803.833	.130562	.718908
2	3	2318.767	72	4803.833	.482691	.695351
12	3	1506.567	72	4803.833	.313618	.815474

STAT. GENERAL MANOVA	Summary of all Effects; design: (dimast2.sta) 1-SEX, 2-PRESENT					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	654.721	12	6879.438	.095171	.762994
2	3	4451.194	12	6879.438	.647029	.599663
12	3	4638.464	12	6879.438	.674251	.584199

Masters results

AT. NERAL NOVA	Summary of all Effects; design: (dimast2.sta) 1-SEX, 2-PRESENT					
fect	df Effect	MS Effect	df Error	MS Error	F	p-level
	1	978.882	12	4786.333	.204516	.659166
	3	3149.857	12	4786.333	.658094	.593334
	3	1658.524	12	4786.333	.346512	.792343

AT. NERAL NOVA	Summary of all Effects; design: (dimast2.sta) 1-SEX, 2-PRESENT					
fect	df Effect	MS Effect	df Error	MS Error	F	p-level
	1	1640.529	12	7010.750	.234002	.637279
	3	2487.492	12	7010.750	.354811	.786588
	3	171.937	12	7010.750	.024525	.994521

AT. NERAL NOVA	Summary of all Effects; design: (dimast2.sta) 1-SEX, 2-PRESENT, 3-PRE/POST					
fect	df Effect	MS Effect	df Error	MS Error	F	p-level
	1*	684.7562*	72*	110.9743*	6.170403*	.015318*
	3	30.0729	72	110.9743	.270990	.846118
	1	.1563	72	25.1465	.006214	.937389
	3	81.6062	72	110.9743	.735362	.534330
	1	6.0062	72	25.1465	.238850	.626523
	3	10.8062	72	25.1465	.429731	.732318
3	3	37.7896	72	25.1465	1.502775	.221122

AT. NERAL NOVA	Summary of all Effects; design: (dimast2.sta) 1-SEX, 2-PRESENT, 3-PRE/POST					
fect	df Effect	MS Effect	df Error	MS Error	F	p-level
	1	111.2426	12	77.38541	1.437514	.253683
	3	21.1528	12	77.38541	.273343	.843480
	1	13.5956	12	15.88542	.855853	.373132
	3	20.6448	12	77.38541	.266779	.848076
	1	.3603	12	15.88542	.022681	.882792
	3	14.2480	12	15.88542	.896924	.470997
3	3	24.2004	12	15.88542	1.523435	.258871

AT. VERAL VOVA	Summary of all Effects; design: (dimast2.sta) 1-SEX, 2-PRESENT, 3-PRE/POST					
ect	df Effect	MS Effect	df Error	MS Error	F	p-level
	1	420.0074	12	205.8438	2.040418	.178675
	3	120.7321	12	205.8438	.586523	.635314
	1	.3603	12	35.9271	.010028	.921885
	3	108.5417	12	205.8438	.527301	.671879
	1	2.6544	12	35.9271	.073883	.790384
	3	14.0655	12	35.9271	.391501	.761340
	3	27.7321	12	35.9271	.771901	.531673